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Explaining the Impact of Social Policy on Child Mortality: A Cross-country Statistical Analysis and a Case Study of Vietnam

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Explaining the Impact of Social Policy on Child Mortality: A Cross-country Statistical Analysis and a Case Study of Vietnam

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A Thesis Submitted for the Degree of Doctor of Philosophy

University of Bath

Department of Social and Policy Sciences

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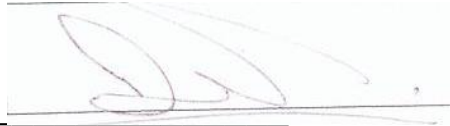
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Abstract

This thesis examines the impact of social policies on child mortality. It argues that structural factors explain most of the variation in child mortality across countries and time. But that in Vietnam the state implemented effective social policies; leading to this country having low child mortality for its structural factors (income, income equality and women's power).

This thesis uses panel data econometrics to investigate the structural determinants of child mortality. Our model shows that national income and women's power reduce, and income inequality increases, child mortality. These independent variables are significant at the 1% level and explain over 90% of the variation in child mortality when our dependent variable is under-five mortality from the World Development Indicators dataset. These results are robust to changes in the functional form, lag structure, dataset and measure of child mortality used in our model. Vietnam is an outlier in our model; it has low child mortality for its structural factors. We consider that Vietnam's effective social policies may explain why it is an outlier.

This thesis also undertakes a detailed case study of Vietnam's social policies. We argue and provide considerable evidence that in Vietnam the government implemented effective family planning, child immunization and female education policies and that these reduced child mortality.

Developing countries are currently committed through MDG4 to reducing under-five mortality by two thirds between 1990 and 2015. Our results show that developing countries are unlikely to achieve this goal because social policies have a small impact on child mortality relative to structural factors.

Acronyms

Acronym	Definition
ADB	Asian Development Bank
CPIA	Country Policy Institutions Assessment
CHC	Commune Health Centre
DHS	Demographic Health Survey
GDP	Gross Domestic Product
GLS	Generalized Least Squares
ICDS	Inter-censal Demographic Survey
IUD	Intrauterine Device
MDG	Millennium Development Goal
MICS	Multiple Indicators Cluster Survey
OLS	Ordinary Least Squares
UNDP	United Nations Development Programme
UNICEF	United Nations International Children's Fund
VLSS	Vietnam Living Standards Survey
WB	World Bank
WDI	World Development Indicators
WHO	World Health Organization

Definition of Terms

This thesis discusses the determinants of child mortality. The term child mortality is not always used consistently in the literature. Some studies use the term child mortality to refer to under-three mortality; while others use this term to refer to under-five mortality. Other studies use child mortality as a general term to refer to infant, under-three and under-five mortality. In addition, many of the multivariate models of under-five mortality reviewed in this thesis are checked for robustness by examining whether they can explain infant or under-three mortality. Other multivariate models may start by discussing under-five or under-three mortality but actually run regressions using infant mortality as the dependent variable.

This thesis uses the term child mortality to refer to infant, under-three and under-five mortality. This is justified because these three measures are normally strongly correlated and much of the multivariate literature specifies models which are tested using infant, under-three and under-five mortality data.

We use the term child survival to refer to neonatal, post neonatal, infant, under-three and under-five mortality. This is necessary because our econometric model in chapter 4 is tested using all these different measures of child survival. Having to write out all five definitions of child survival each time we wanted to refer to this model would be cumbersome.

The table below shows the terms we use to refer to different measures of child survival.

Term	Definition
Neonatal Mortality	The probability of dying within the first month of life; expressed per 1000 live births
Post Neonatal Mortality	The probability of dying between the second and 12 th month of life; expressed per 1000 live births
Infant Mortality	The probability of dying in the first year of life; expressed per 1000 live births
Under-three Mortality	The probability of dying in the first three years of life; expressed per 1000 live births
Under-five Mortality	The probability of dying in the first five years of life; expressed per 1000 live births
Under-ten Mortality	The probability of dying in the first ten years of life; expressed per 1000 live births
Child Mortality	We regard infant, under-three and under-five mortality as specific measures of child mortality
Early Child Mortality	We use this term to refer to neonatal and post neonatal mortality
Child Survival	We use this term to refer to child and early child mortality

Chapter 1: Introduction and Overview of Methodology

Introduction to Research Question

Death is inevitable. But the death of children due to a failure by governments to spend their resources wisely and implement effective social policies is a tragedy. The MDGs recognize this, with the fourth goal committing the governments of developing countries to reducing under-five mortality by two thirds between 1990 and 2015.

Most developing countries are not on course to achieve this goal. A detailed empirical analysis by Murray et al (2007) predicts trends in under-five mortality for nearly all developing countries. This study concludes that only 28 middle-income and four low-income countries have a greater than 75% chance of achieving MDG4. A more recent study by You et al (2010) calculates that of the 67 countries with the highest under-five mortality rates only 10 are on course to have reduced under-five mortality by two thirds between 1990 and 2015. And our own statistical analysis concludes that out of 61 countries where reliable and comparable mortality data exist only 22 at most¹ are currently on course to achieve MDG4.

Three sources of evidence suggest that MDG4 was ambitious, but not impossible to achieve. First, the already low levels of child mortality in some developing countries such as Sri Lanka and Vietnam imply that other developing countries, if they implemented similar social and economic policies, could sharply reduce child mortality and achieve the fourth MDG. Second, the high rates of decline in child mortality in Vietnam, Bangladesh and Nepal² also illustrate that it is not impossible for developing countries to achieve MDG4. And third, most children in developing countries die from diseases for which well known, proven and cost effective health interventions exist (Jones et al 2003 and Cutler et al 2006).

Why are most countries not currently on course to achieve MDG4? It could be because their governments are not implementing best practice social policies. Yet this seemingly simplistic statement hides three complex questions. First, to what extent are changes in child mortality driven by social policies as opposed to structural factors like economic growth, income inequality and women's power? Second, what are effective social policies for reducing child mortality? And third, what underlies a state's ability to implement effective social policies?

This thesis answers these questions. It does so using the following methodology. First, we ask - to what extent are changes in child mortality driven by social policies as opposed to structural factors? Panel data econometrics is used to determine the importance of structural variables in explaining child mortality across countries and time. We then use this panel data econometric model to select an outlying country (Vietnam) which has low child mortality for

¹ Our analysis in appendix 13 shows that 22 developing countries are on course to achieve MDG4 when the linear functional form is used. The linear-log and log-linear functional forms show two and zero countries achieving MDG4 respectively.

² As shown in our later econometric analysis, and by Murray et al (2007) and You et al (2010).

its structural factors. Our underlying assumption is that Vietnam's low child mortality (for its structural factors) should be explained by the efficacy of its social policies.

This thesis examines the social policies the Government of Vietnam used to reduce child mortality. It argues that the government implemented effective social policies in the areas of female education, child immunizations, child healthcare and family planning. These policies were effective compared to other countries with a similar income and explain why Vietnam has low child mortality for its structural factors. We also briefly discuss the state institutions that underlay the implementation of these social policies and the historical development of a powerful state in Vietnam.

The structure of this thesis is as follows. Chapter 2 reviews the multivariate literature on the determinants of child mortality. Chapter 3 constructs a detailed analytical framework of the determinants of child mortality to guide our econometric model. Chapter 4 tests the hypotheses outlined in the analytical framework using a panel data econometric model. Chapter 5 analyzes child mortality and social policy in Vietnam. And chapter 6 summarizes our arguments and discusses their policy implications.

Overview of Each Chapter

Chapter 2 identifies the socioeconomic determinants of child mortality. It does this by reviewing the previous literature on panel data and cross-country multivariate models of child mortality. This literature review concludes that income, income inequality, female education and health outputs determine most of the variation in child mortality across countries and time. Many different multivariate studies using different datasets, functional forms and estimation techniques have consistently found that these variables are significant determinants of child mortality.

Many multivariate studies can, however, be criticized for using data from international databases such as the WDI. The problem with data from these datasets is discussed in greater detail in chapter 2. Existing multivariate studies can also be criticized as most do not contain a detailed analytical framework showing the causal pathways between different socioeconomic variables and proximate variables. The lack of an analytical framework makes it difficult to derive meaningful policy recommendations from many multivariate studies. For example, Hanmer et al (2004) and Easterly (1999) argue that national income per capita improves child health. If national income is reducing child mortality solely through its association with increased family income then the policy conclusion would be that increased social expenditure is not needed to achieve MDG4. Yet if national income is reducing child mortality solely through increased social spending then a reasonable policy conclusion would be that more social spending is required to achieve MDG4. Our panel data model overcomes these problems because it is based on a detailed analytical framework and an accurate dataset.

Our literature review also highlights that there is an ongoing debate regarding the effectiveness of government health expenditure in reducing child mortality. Three sources of evidence suggest that government health spending can reduce child mortality. First, most child deaths occur from a few diseases for which well known, proven, cost effective public health interventions exist (Jones et al 2003 and Cutler et al 2006). Second some multivariate studies find that immunizations and medical personnel are associated with lower mortality (Wang 2002 and Anand and Baernighausen 2004). And third some government programmes

in developing countries have substantially reduced child mortality (Victora et al 1996 and Gutierrez 1994). Yet multivariate studies that directly estimate the impact of government health expenditure on child mortality conclude that it has an extremely small impact (Filmer and Pritchett 1999).

We can explain this paradox by examining the methodologies used by epidemiological, health intervention, programme studies and multivariate studies.

Epidemiological studies examine the number of children dying from particular diseases. Such studies find most children die from curable, well understood causes for which proven health interventions exist, they conclude that increased health provision, and presumably spending, could rapidly reduce child mortality.

Health intervention studies build on epidemiological studies by explicitly costing these proven health interventions. The problem with this methodology, from the viewpoint of examining health expenditures actual effectiveness, is that it implicitly assumes that all additional public health expenditure will be aimed at efficiently reducing child mortality. However, some developing countries' governments may use additional expenditure for the purpose of political patronage or inefficiently spend it on prestigious tertiary healthcare facilities (Peters et al 1999). They may also lack the capacity to rapidly scale up effective interventions (Schell et al 2007). Health intervention studies, therefore, show the potential effectiveness of government expenditure across a range of interventions.

Some studies also conclude that specific government programmes have effectively improved child mortality. Gutierrez (1994) argues that the national programme to reduce diarrheal diseases in children in Mexico reduced child mortality. The fact that health expenditure can potentially, and in a few countries has, effectively reduced child mortality does not necessarily imply that on average across all countries health expenditures have significantly reduced child mortality.

Cross-country regressions measure the average effectiveness of government expenditure across all nations. This can lead to the conclusion that health expenditure cannot reduce child mortality - an erroneous conclusion based on the methodological error of confusing the existing average relationship with the potential relationship. The regression has shown that on average across all countries expenditure has not reduced child mortality: not that government spending cannot potentially reduce child mortality.

So, in summary, our reading of the evidence from epidemiological, health intervention, multivariate and programme studies is that it shows government health expenditure can potentially, and in some countries has, effectively improved child survival, but that on average across countries government health expenditures actual impact has been low. This suggests that there may be a subset of countries where effective government health and social spending have reduced child mortality. Chapter 5 of this thesis builds on this point by examining if social policy in Vietnam can explain why this country has low mortality for its structural factors.

Chapter 3 constructs an analytical framework of the causes of child mortality. This framework shows how socioeconomic variables determine mortality through proximate variables. It demonstrates how income, income inequality and the power of women in society affect mortality through proximate variables such as birth spacing. This framework also

illustrates how state capacity and politics determine the effectiveness of public expenditure in reducing mortality through health outputs, female education and water supply and sanitation. We only included socioeconomic variables in this framework if there was considerable empirical and theoretical evidence from our literature review that they were determinants of child mortality.

Testable hypotheses were also derived from this framework. The most important of these are: a) income, income inequality, and women's power should explain the majority of the variation in child survival and under-nutrition; b) that income inequality should affect child mortality through the aggregation hypothesis (diminishing marginal returns) and c) public expenditure should have a more significant impact on child mortality in high capacity states.

Chapter 4 tests these hypotheses using panel data econometrics. Our analysis uses a different dataset from previous studies. Most previous studies used data from UNICEF and/or WDI. These should ideally be based on data from all relevant countries' national statistics agencies. These agencies should in turn have collated this data from a comprehensive and accurate vital registration system of all births and deaths. Yet most developing countries lack the necessary systems to collect data on all child births and deaths. The cross-country databases provided by international organizations are therefore based on data collected via vital registration systems, national censuses, multiple indicator clusters and DHS (Murray et al 2007 and Wang 2002). This leads to two problems. First, different sources give different estimates of under-five mortality, suggesting that data from different sources may not be strictly comparable across countries or periods³. Second, for some countries and years actual data does not exist, so the reported under-five mortality rate is itself predicted based on past trends (Murray et al 2007). As a result of this, time series and panel data analysis based on these data is likely to predict trends which are determined by the rules and assumptions governing the interpolations and not the actual rate of change in under-five mortality (Bhargava et al 2001).

DHS provide an alternative source of child mortality data. These surveys are accurate and comparable across countries, but are undertaken at irregularly spaced intervals and do not provide yearly estimates of child mortality (Sahn and Stifel 2003). The five year average rates of child mortality calculated by Macro-International from DHS may also be biased downwards. The reason is that the average age of children's mothers, which is a significant determinant of child mortality, will decrease as mortality is calculated for earlier periods (Sahn and Stifel 2003). We avoid this bias by creating our own dataset by recalculating infant and under-three mortality rates from individual mothers' responses to DHS in 61 developing countries. Our technique gives us seven years of under-three mortality data and nine years of infant mortality data for each survey. These data are comparable both across countries and across time. This dataset is not publicly available and was constructed specifically for our regression analysis. This dataset is arguably the most accurate available. The use of this dataset means our regression analysis offers significant value added over previous multivariate models.

The econometric analysis conducted in chapter 4 provides striking evidence that income, income inequality and women's power determine the majority of the variation in child mortality across countries. The robustness of these results was confirmed using different estimation techniques, functional forms, lag structures and measures of child survival.

³ See appendix 3 for our own statistical analysis of WDI and DHS datasets

Chapter 4 also argues that governments can only partially control these independent variables in the short term. And that when governments pledged to achieve MDG4 they were principally committing themselves to improved health and social policies and not higher rates of economic growth or lower income inequality. Recently developing countries' governments also committed themselves to the Global Strategy for Women's and Children's Health (UN 2010), which emphasizes achieving MDG4 through health policies and not increased economic growth or reduce inequality.

Our econometric model also found, as did a study by Filmer and Pritchett (1999), that government expenditure on health reduced under-five mortality but not by very much. Yet as discussed earlier, other evidence suggests that social policies can potentially reduce mortality. The conundrum is why government expenditure can potentially, but on average is actually not, effectively reducing child mortality. Differences in state capacity and social policies between countries seem likely explanations. Our econometric analysis, however, provides only weak evidence that the effectiveness of public expenditure in reducing under-five mortality varies with state capacity. The most likely explanation for this is that the interaction between state capacity and the effectiveness of public expenditure is inherently complex and difficult to measure. This relationship does not lend itself to the kind of reductionist criteria needed to undertake a regression analysis. A case study methodology allows us to examine social policies and state institutions in detail in a specific country.

Chapter 4 also identifies countries that are outliers in regressions where measures of child mortality were the dependent variables and income, income inequality and women's power were independent variables. Vietnam was consistently found to be an outlier in regressions with different lag structures, measures of child mortality, estimation techniques and functional forms. We conclude chapter 4 by arguing that Vietnam's low child mortality for its structural factors may be explained by its social policies.

Chapter 5 of this thesis undertakes a case study of child mortality and social policies in Vietnam. This chapter is divided into five main parts. The first part reviews available sources of data on child mortality and its determinants in Vietnam. This includes a detailed discussion of the advantages and disadvantages of data from VLSS, MICS and DHS.

The second part reviews existing studies of the determinants of child mortality in Vietnam, many such studies were undertaken by international organizations such as the WB, ADB, UNICEF and WHO. This review shows that there is some evidence that family income, mother's education, access to clean water and sanitation and health outputs are important determinants of the variation in child mortality across families in Vietnam.

The third part reviews the distribution of child mortality by income, region and ethnicity in Vietnam. Inequality in the distribution of child mortality by income in Vietnam increased during the 1990s and is now as unequal as in many other developing countries. Many health outputs, such as vaccinations, are also as unequally distributed in Vietnam as in other developing countries. Vietnam's low child mortality for its structural factors is not explained by a more equal distribution of health outputs leading through diminishing marginal returns to lower overall child mortality.

There is also significant variation in child mortality across regions in Vietnam. Our analysis suggests that poverty and access to vaccinations are the most likely determinants of under-five mortality across regions.

In Vietnam, ethnic minorities have a higher rate of child mortality than the majority of the population. Our analysis suggests that this is because ethnic minority groups are poorer, have lower levels of education, worse access to clean water and sanitation, higher fertility and worse access to high quality health services than the majority of the population.

Part 4 is the longest and most detailed part of chapter four. It examines whether specific social policies implemented by the government can explain why Vietnam has low child mortality for its structural factors. It shows that Vietnam has effective social policies. More specifically, it provides considerable evidence that policies in the areas of primary healthcare, child immunizations, female education and family planning were more effective in Vietnam than in other countries with a similar income. There is much statistical evidence to support this argument and most commentators also agree that the Government of Vietnam's social policies have effectively reduced child mortality.

Part 5 argues that the Government in Vietnam has developed specific institutions such as mass organizations and an intertwined party and state structure to cooperate with, control and provide services to its citizens. The government has through these institutions implemented effective family planning, child immunizations and women's education policies. Such policies have reduced child mortality.

Discussion of Methodology

The methodology outlined in this thesis may seem complex and unnecessary. Some might even say it involves: "using a sledge hammer to crack a nut". There are broadly speaking two alternative methodologies that this thesis could have followed. First, we could have examined social policies solely through a case study methodology. Second, we could have used econometrics to examine the relationship between countries' social policies, state capacity and child mortality.

We consider that only using the case study methodology would have been problematic. The reason for this is that case studies often find what their authors seek. Formally the case study methodology, when used in isolation, often fails to account for the impact of structural factors and is intrinsically biased towards attributing changes in outcomes to policy variables. Say we were to choose a developing country and start examining its social policies. We could observe a downward trend in child mortality rates and attribute this to changes in government social policies. Yet such changes in mortality could well be due to changes in family income caused by economic growth. This is not just a theoretical point. Many case studies have failed to account for structural factors properly.

Alternatively, we could have examined the effectiveness of social policies exclusively through an econometric methodology. Yet the argument we make throughout this thesis is that the efficacy of social policy is determined by a complex interaction of a range of factors. It is not possible to fully model such a complex interaction through econometrics both because there are limited and inaccurate statistical data on some government social policies and because such a complex relationship does not lend itself to the kind of reductionist criteria needed to include variables in a regression analysis.

Our use of two separate methodologies – econometrics and case study – does mean that the space allocated to each method is more limited than it would be in a thesis which concentrated exclusively on one method. But we consider that the more limited space

allocated to each method is justified because using both methods allows us to control for structural factors and simultaneously carry out a detailed and nuanced analysis of Vietnam's social policies.

The methodology used by this thesis is not of course immune from criticism⁴. One potential criticism with our method is that the unexplained variation in the regression model may not be due to social policy but may instead simply be unexplainable random variation. It is, however, important to realize that we regard the econometric component of our methodology as providing a strong indication, but not absolute proof, that Vietnam has effective social policies. We are saying: our econometric analysis leads us to consider that Vietnam has effective social policy and does other evidence support this? Do government and donor evaluations conclude the Vietnam's social policies have reduced child mortality? Do other academic studies conclude that Vietnam has effective social policies?

In conclusion, this thesis examines the impact of social policy on child health. It first constructs an econometric model which is used to select a country (Vietnam) which has low child mortality for its structural factors. The second part of this thesis examines how Vietnam's social policies have reduced child mortality.

⁴ For a more technical description of the potential problems with this methodology see appendix 1.

Chapter 2: Literature Review

Introduction

This chapter identifies the socioeconomic determinants of child mortality. First, we review the study by Cutler et al (2006) on the determinants of mortality and based on this we specify a few salient categories of determinants. Second, we undertake a detailed review of multivariate studies to find the specific determinants of child mortality. Third, chapter 3 arranges the socioeconomic determinants of child mortality in an analytical framework. This framework demonstrates how (the causal pathway through which) each relevant socioeconomic variable affects mortality. These causal pathways are, to avoid repetition, not discussed in detail in this chapter. The third chapter tests this analytical framework using panel data econometrics.

Cutler et al (2006) undertake a seminal review of the determinants of mortality. This paper examines the determinants of mortality across time, between countries and within countries. Although it concentrates on examining mortality it is still highly relevant to our discussion of child mortality. The reason is that much of the difference in mortality across countries and time is due to differences in child mortality and therefore many of the determinants of mortality identified by this paper may also be determinants of child mortality.

According to Cutler et al (2006) the largest declines in mortality seen in England and Wales and France in the 18th century and in the USA from 1790 were concentrated among younger age groups and occurred due to a sharp reduction in infectious diseases. They state that income (through improved nutrition), better public health (mainly improved water and sanitation), urbanization, vaccinations and other medical treatments are possible determinants of this decline in mortality. This paper concludes that Britain and France experienced three phases of mortality reduction through history. In the first phase from the middle of the 18th century to the middle of the 19th century improved nutrition and economic growth may have played a leading role in reducing mortality. In the second phase, which lasted from the end of the 19th century into the 20th century, clean water and sanitation and knowledge of personal health interventions drove reductions in mortality. In the third phase, dating from 1930 onwards, vaccinations, antibiotics and other healthcare interventions led to mortality decline.

Cutler et al (2006) also examine explanations of differences in child mortality between countries. They note that much of the difference in mortality between rich and poor countries is due to under-five mortality and that more deaths are from infectious diseases in developing countries. The main socioeconomic determinants that have been put forward as explanations for these differences in mortality are income per capita, the disease environment (mainly determined by water supply and sanitation), public health interventions (particularly child immunizations) and women's education.

One critical point they make is that the high rates of mortality in many poor countries are not primarily due to a lack of suitable treatments. Diarrhoeal diseases and respiratory infections, the first and fourth leading causes of death worldwide, are easily treatable with oral rehydration therapy and antibiotics respectively. Both these treatments are cheap and cost-effective compared to many other health interventions. Other infectious diseases such as whooping cough, tetanus, polio, diphtheria and measles kill more than a million children

under four each year in developing countries but have been eliminated in western countries through near universal immunization programmes. The large number of deaths due to easily curable diseases for which cost-effective interventions exist suggests that government capacity may be an important determinant of the effectiveness of health interventions and mortality.

Cutler et al (2006) also conclude that the role of income in explaining differences in mortality across countries may have been over emphasized. They argue that for India and China there is a negative correlation between decadal rates of economic growth and progress in child mortality. China's remarkable post-war reduction in infant mortality happened prior to the acceleration of economic growth in the 1980s. This paper, therefore, argues that income may have a limited impact unless it occurs with important public health measures which require political will and do not automatically occur with economic growth.

This paper also examines the determinants of mortality within countries. It notes that within countries there is a relationship between income and health. These inequalities in health may be due to access to healthcare, health related behaviours (such as smoking) and/or a lack of social status and related stress.

This seminal article informs the analysis contained in this thesis in four ways.

First, we arrange our literature review around the following salient groups of variables - income, education, health interventions, spending and government capacity, water supply and sanitation and proximate variables. These groups are very similar to, and are based on, the main determinants of mortality identified by Cutler et al (2006) across time and countries.

Second, they argue that income may affect mortality through nutrition. Similarly our analytical framework in chapter 3 discusses the relationship between nutrition and child mortality in detail and in chapter 4 we test whether our models of child survival can also explain differences in under-nutrition.

Third, they emphasize that cost-effective health interventions exist for many diseases which cause child mortality, but that some governments may lack the political will or capacity to implement these health interventions. This thesis builds on this argument by explicitly including government capacity in its analytical framework and testing whether the effectiveness of public spending in reducing child mortality varies with state capacity using econometrics. We also discuss the impact of state capacity on social policies in chapter 5.

Fourth, they argue that China succeeded in reducing child mortality prior to experiencing strong economic growth. This supports our argument that while structural factors may explain the majority of variation in child mortality across countries and time, there may be a subset of countries where amenable politics and high state capacity have meant social policies have driven reductions in child mortality.

Table 1 shows the number of studies which found each determinant of child mortality to be significant organized according to the salient groups we defined earlier. Our review concentrated on panel data and cross-country multivariate models. Models which explain variation in mortality across families from many different countries are also reviewed and included in table 1. We do not discuss time series multivariate models that focus on a particular country because we are not principally interested in location specific determinants

of child mortality. Likewise, we do not review studies which examine differences in mortality solely in developed countries as this thesis concentrates its argument on the developing world. Classifying variables by determinant is not an exact science because some studies include many different models and there is a degree of leeway in defining different categories of variables. Nevertheless, table 1 can be interpreted as showing the approximate empirical support for the different socioeconomic determinants of mortality.

There are two further problems with the results displayed in table 1. First, it places equal weight on all multivariate studies. Yet some studies use superior statistical techniques and data compared to others. Second, this table says nothing about the strength of the theoretical argument linking the different independent variables to child mortality.

For this reason this chapter discusses in detail each salient group of variables in turn. More specifically, we discuss income (GDP per capita and GNP per capita), education (female education and male education), healthcare (health interventions, public spending on healthcare and state capacity), environmental factors (water supply and sanitation) and proximate variables. We also discuss, in a separate section, how the functional forms, econometric techniques and datasets used in the multivariate studies reviewed influence the econometric analysis we undertake in chapter 4.

A more detailed literature review is available to the reader on request. This detailed literature review separately discusses the methodology, datasets, conclusions and problems with each of the 30 separate multivariate models. Table 1 in appendix 2 shows the source of data, sample size, adjusted R-Squared, dependent variable and independent variables for each of the 30 studies referenced below.

Table 1: Summary of Multivariate Studies Reviewed

Salient Variable Groups	Determinant of Child Mortality ⁵	Number of Studies Significant in
Income	GDP or GNP per capita ⁶	24
	Income Inequality ⁷	12
Education	Female Education or Literacy ⁸	13
	General Education or Male Education ⁹	9
Healthcare	Health Interventions ¹⁰	9
	Public Health Expenditure ¹¹	3
	State Capacity ¹²	3
Environmental	Access to Water Supply or Sanitation ¹³	4
Proximate Variables	(Birth Order, Birth Spacing and Mother's Age at Birth) ¹⁴	4

⁵ The table only shows those variables that were found to be significant by four or more multivariate studies. Other independent variables which were found to be significant determinants of child mortality by less than four but more than one multivariate study included (with the number of studies they were found to be significant in in brackets): measures of gender disparity (3), ethnic linguistic fractionalization (3), energy consumption (2), rural or urban location (2), located in the tropics or not (2) and whether a country was Muslim or not (2). Independent variables which were only found to be significant in one study are not shown.

⁶ Anand and Baernighausen (2004); Asafu-Adjaye (2004); Shen and Williamson (1999); Easterly (1999); Filmer and Pritchett (1999); Flegg (1982); Franz and Fitzroy (2006); Hanmer et al (2003); Heaton et al (2005); Jamison et al (2006); Lazaroova (2006); Fay et al (2003); McGuire (2006); Pritchett and Summers (1996); Ram (2006); Rodgers (1979); Shandra et al (2005); Singh (1994); Waldmann (1992); Wang (2002) and Younger (2001).

⁷ Rodgers (1979); Waldmann (1992); Rajkumar and Swaroop (2007); Fay et al (2003), Asafu-Adjaye (2004); Franz and Fitzroy (2006); McGuire (2006); Ram (2006) and Schell et al (2007). Note Ram runs four separate models all of which find income inequality significant.

⁸ Flegg (1982); Hobcraft et al (1985); Singh (1994); Shen and Williamson (1999); Filmer and Pritchett (1999); Rajkumar and Swaroop (2007); Fay et al (2003); Heaton et al (2005); Ram (2006); Franz and Fitzroy (2006); McGuire (2006) and Schell et al (2007).

⁹ Shen and Williamson (1999); Asafu-Adjaye (2004); Desai and Alva (1998); Pritchett and Summers (1996); Ram (2006); Rutstein (2000); Shandra et al (2005) and Younger (2001).

¹⁰ See: Anand and Baernighausen (2004); Shen and Williamson (1999); Hanmer et al (2003); Heaton et al (2005); Fay et al (2003); Rutstein (2000); Singh (1994); Wang (2002) and Younger (2001).

¹¹ Filmer and Pritchett (1999); Wang (2002) and Schell et al (2007).

¹² Shen and Williamson (1999); Lazaroova (2006) and Schell et al (2007).

¹³ Desai and Alva (1998); Fay et al (2003); Rutstein (2000) and Wang (2002).

¹⁴ See Hobcraft et al (1985); Rutstein (2000), Fay et al (2003); Heaton et al (2005) and Franz and Fitzroy (2006).

National Income

Many multivariate studies have found that national income per capita and income inequality at the national level, are associated with child mortality.

GDP and National Income Per Capita

There is much evidence that high national income per capita is associated with lower child mortality. The majority of econometric studies find that some measure of income per capita (e.g. GDP per capita) is a significant determinant of child mortality. The academic consensus on this point means that while most studies include some measure of income per capita as an independent variable, they concentrate their argument on proving the existence of a relationship between child mortality and some other less researched variable. This section, therefore, discusses studies by Pritchett and Summers (1996) and Easterly (1999) that concentrate on examining the relationship between income and child mortality.

Pritchett and Summers (1996) test the impact of income on infant and under-five mortality. They measure income using GDP per capita. This paper uses a panel dataset that includes observations from different developing countries and for different years. Their model specifies that the log of infant or under-five mortality is determined by the log of income, other variables, country fixed effects and time fixed effects. The inclusion of country fixed effects allows for the presence of any number of unspecified country-specific, time invariant variables that could affect infant or under-five mortality. The inclusion of time fixed effects allows infant and under-five mortality to shift downward or upward over time. Time fixed effects are included to account for exogenous technological change.

This paper finds that the estimated long run elasticity between income and infant mortality and under-five mortality is between -0.2 and -0.4 in developing countries. Using these estimates, the authors calculate that over half a million child deaths in the developing world in 1990 alone can be attributed to the poor economic performance in the 1980s. They also argue that differences in economic growth rates between countries explain approximately 40% of the variation in infant and under-five mortality.

Pritchett and Summers (1996) test for the direction of causality between income and infant mortality using an instrumental variable technique. This involves replacing income in their model with instruments. These are variables that could cause economic growth but have no relationship with infant mortality and are not associated with any third factor which could cause changes in both infant mortality and income. The authors argue that terms of trade shocks, ratio of investment to GDP and the deviation of the official exchange rate from its purchasing power parity level fulfil these conditions and are valid instruments.

This paper shows that when income is replaced with the investment ratio, the coefficient on the investment ratio is significant. The other instruments are, however, statistically insignificant when they are used instead of income. When the paper combines all the instruments, and all the instruments other than the deviation of the official exchange rate from its purchasing power parity level, the coefficient on the combined instruments variable is higher than the original coefficient on GDP and is statistically significant. This paper argues that these results show the instruments are affecting infant mortality and that this can

only be through economic growth. As infant mortality cannot be reasonably assumed to determine the instruments, this provides evidence that economic growth causes improvements in infant mortality.

Pritchett and Summers (1996) attempt to empirically demonstrate that the direction of causality runs from income to health is commendable. The empirical evidence they provide is, however, problematic because the coefficients on some of the instruments are larger than the coefficient on income. For example, the investment ratio explains more of the variation in infant mortality than income does. This is problematic because if the investment ratio works through economic growth then its impact on infant mortality should be smaller than that of incomes: the coefficient on investment can only be larger than the coefficient on income if it is either directly reducing infant mortality or is operating through some unnamed third variable. In either case, it would not be a valid instrument. As the investment ratio includes investments in health facilities then it could be directly reducing infant mortality.

This paper's statement that its instruments are robust determinants of economic growth is also questionable. For although some endogenous growth papers have identified terms of trade shocks, the investment ratio and the deviation of the official exchange rate from its purchasing power parity level as causes of economic growth, other papers have found them to be insignificant determinants of economic growth (Fine 2000).

Pritchett and Summers (1996) aim to test the overall impact of income on health. This means that their model should not include any independent variables which income may determine health through. This paper recognizes this point, arguing that it would be wrong to include public expenditure on health in their model, as this would lead to the coefficient on income showing the partial effect of income on health. Yet their final model includes years of schooling, despite that fact this may be partially determined by income through increased public expenditure on education.

Overall Pritchett and Summers (1996) show that there is a strong relationship between income and child mortality. The instruments they use to prove that the direction of causality runs from income to health may, however, be incorrectly specified as they seem to have a larger impact on child mortality than income and may not be robust determinants of economic growth.

Easterly (1999) examines the relationship between per capita income and infant and child mortality. This paper regresses infant and child mortality against per capita income using fixed effects and first difference models using panel data. It concludes that 58% of the variation in infant mortality and 78% of the variation in child mortality can be explained solely by per capita income. This model is clearly simplistic because it excludes variables such as female education that could explain the cross-country variation in infant and child mortality. It does, however, serve to emphasize the importance of per capita income in explaining the cross-country variation in infant and child mortality.

In conclusion, Pritchett and Summers (1996) and Easterly (1999) provide strong evidence that increased income per capita at the national level is associated with lower child mortality. Many other empirical studies also find that GDP per capita or GNP per capita are important determinants of child mortality across countries and time. This section has not discussed how income at the national level increases mortality; for instance whether it is through increased

private consumption or because richer countries spend more on healthcare. These causal pathways are, however, discussed in more detail in the next chapter.

Income Inequality

Income inequality is associated with high infant and child mortality across countries and time. Multivariate studies support this statement with 12 of the 30 models reviewed finding that income inequality is a significant determinant of mortality. This section reviews studies by Rodgers (1979), Asafu-Adjaye (2004), Beckfield (2004), Ram (2006) and Waldmann (1992), all of which concentrate on examining the relationship between income inequality and child health.

Rodgers (1979) was the first to examine the relationship between income inequality and child mortality. This paper argues that income and income inequality are of overriding importance in describing changes in child mortality during development. Income is important both because it directly improves child mortality by increasing consumption and because it drives, or is strongly correlated with, other determinants of child mortality such as the provision of health services. This paper also argues that income reduces infant and under-five mortality but by at an ever-decreasing rate.

This paper provides empirical evidence to support its hypothesis that income inequality increases, and income reduces, infant mortality. More specifically, it finds that both 1/GDP per capita (showing diminishing marginal returns to income) and the Gini coefficient (showing income inequality) are significant determinants of infant mortality and under-five mortality.

One criticism of this study is that its dataset is now some 30 years out of date and that the sample size is relatively small ($n = 56$). Ram (2006) re-estimates Rodgers' (1979) model with a larger, more accurate dataset which contains observations until the late 1990s and finds that the original results are well replicated with income significantly decreasing, and income inequality significantly increasing, infant mortality.

A further criticism of Rodgers' (1979) model is that it excludes variables that are robust determinants of infant and child mortality across countries, such as female education. However, in its defence, this paper does explicitly state that it is seeking to describe the relationship between development and child mortality: not find all the significant determinants of infant and under-five mortality.

Asafu-Adjaye (2004) investigates the relationship between income inequality and infant mortality. This study finds, using a panel dataset including data from 1970 to 1995, that income inequality, savings and education are statistically significant determinants of infant mortality. Income inequality is measured using the Gini coefficient; savings are measured as the proportion of total savings to GDP and education is measured by the ratio of total enrolments in primary school to the population aged between 15 and 65 years of age. Education and savings reduce, and income inequality increases, infant mortality. Savings are used as a proxy for the capacity to improve healthcare. This paper also divides its dataset into low and high-income countries and reruns the regression; these results show that the relationship between income inequality and health is stronger in low-income countries. It

concludes that income inequality has a significant effect on infant mortality in developing countries.

However, not all studies conclude that income inequality increases child mortality. Beckfield (2004) argues that studies that find a statistically significant relationship between income inequality and child mortality use small datasets and inappropriate controls. This paper re-examines this relationship using a larger sample (692 observations from 115 countries between 1947 and 1996) and a wider range of statistical controls, including fixed effects models that address heterogeneity bias. This study concludes that when fixed effects models are used the association between income inequality and infant mortality disappears.

The studies discussed above show that income inequality is associated with higher child mortality, but they do not investigate how income inequality determines child mortality. Three different causal pathways between income inequality and infant mortality can be identified.

First, income inequality may be associated with child mortality because there are diminishing marginal returns between income and child mortality at the family level. Increasing 100 poor families' incomes from \$400 to \$450 may reduce infant mortality by more than increasing a single family's income by \$5000. Under both these scenarios the country's GDP per capita would increase by the same amount but under the second scenario income inequality would be higher. At the national level, this implies that for a given income, the more unequal the mean deviation measure of income dispersion between families the higher the overall rate of infant and child mortality. Because different measures of income inequality are highly correlated, most measures of income inequality can be substituted for the mean deviation measure of dispersion. We label this causal pathway the aggregation hypothesis.

Second, income inequality could cause higher child mortality through the psychosocial mechanism or reduced social capital. The psychosocial effects mechanism postulates that humans are envious creatures whose health suffers due to stress or related factors when they realize that they have a low income or position in society compared to others. Academics originally proposed this mechanism to explain the link between income inequality and life expectancy (Wilkinson 1996). It can only explain the link between income inequality and child health if either children are aware of their social position, or else if their parents' knowledge of social status partly determines child mortality. The applicability of this mechanism to developing countries where most children die from non-stress related infectious diseases is debatable.

The social capital mechanism states that income inequality extenuates actual or perceived differences in interest across individuals. This reduces trust, hampering cooperation and ultimately leading to government policies that only provide basic services to certain groups; increasing infant and child mortality. It may also lead to an unjust social climate, limiting access by the poor to goods and services and further reinforcing income inequality.

Third, income inequality could be acting as a proxy for a third factor such as the power of different socioeconomic groups in society to influence government policy.

Waldmann (1992) develops a methodology to test how income inequality causes or is associated with child mortality. In this model infant mortality is the dependent variable and the independent variables are share of the income controlled by the poor and the share of

income controlled by the rich. This paper finds that both variables are significant and that the coefficient on the income share of the rich is positive. That is holding the absolute income of the poor constant, infant mortality increases when the share of national income controlled by the richest five percent of society increases. This result suggests the aggregation hypothesis is incorrect, because income inequality increases infant mortality even when the poor do not see their actual income fall.

Despite these results, Waldmann does not conclude that income inequality causes infant mortality to increase. He instead argues that a third factor is associated with both an unequal income distribution and higher infant mortality. His paper examines provision of medical services, the degree of urbanization, the extent of female literacy and differences in the composition of births among groups, but it does not find any evidence that these explain income inequality or infant mortality. The paper concludes that the association between income inequality and child health occurs either because a larger fraction of babies are born to poor families in countries where the rich capture a large share of income or because government policies drive both income inequality and infant mortality. The argument that government policy determines income inequality and infant mortality appears plausible. However, this paper's results could also be explained by income inequality causing, through the psychosocial or social capital causal pathways, poor children to die. Waldmann (1992) does not consider these causal pathways, presumably because he regards them as implausible.

To summarize, there is strong empirical evidence that income inequality is associated with, or causes, high child mortality across countries. There is, however, still some debate over the mechanism through which income inequality causes, or is associated with, child mortality.

Education

Many multivariate studies have found that female education reduces child mortality across countries and time. Others have found that education in general determines child mortality. We discuss studies which examine the relationship between these two variables and child mortality in detail in the next two sections.

Female Education

There is much evidence that female education reduces child mortality. More specifically, of the 30 multivariate studies we reviewed, 13 found that female education affects child mortality. This section discusses four studies - Flegg (1982), Subbarao and Raney (1993), Singh (1994) and Schell et al (2007) that concentrate on determining the relationship between female education and child mortality.

Flegg (1982) aims to explain why some countries have poorer child health than others. Following Caldwell (1979), this paper argues that increasing women's education would reduce infant and child mortality through three mechanisms. First, educated mothers may become less fatalistic and adopt alternative, more modern, measures of childcare that become available as countries develop. Second, an educated mother is more capable of demanding the attention of health professionals. Third, educated mothers may ensure their children receive a larger share of household resources.

This paper includes the log of infant mortality as the dependent variable and the significant independent variables are the Gini coefficient, female illiteracy, the number of physicians per 1000 people and the number of nurses per 1000 people. GDP per capita is not found to be a significant explanatory variable. This paper shows that these results are robust to the exclusion of the richest developing countries from the dataset and the inclusion of developed countries.

Most multivariate studies find that income reduces infant mortality. Yet Flegg (1982) concludes that income has no significant effect on infant mortality. This result could be occurring because this model is incorrectly specified and only tests the partial impact of income. More specifically income may partially determine the amount of social expenditure. This may in turn determine the number of doctors and nurses. This paper's result can then be interpreted as showing that income may not directly determine infant mortality.

Subbarao and Raney (1993) examine the social gains from female education. This paper argues that increased female education could reduce infant mortality through better use of available health facilities and by increasing mothers' knowledge of hygiene, sanitation and healthcare. Using a dataset of 70 developing countries, they find that the female gross secondary school enrolment rate, family planning services, population per physician, GDP per capita and a Latin American regional dummy variable are significant determinants of infant mortality.

This paper shows that increasing female education could substantially reduce infant mortality. According to its model, doubling female secondary school enrolments in 1975 from a mean of 19% to 38% would more than halve infant mortality: while doubling GDP per capita would only decrease infant mortality by approximately 6%. The authors conclude that increasing female secondary school enrolment would help reduce infant mortality.

This model may, however, be incorrectly specified. The reason for this is that higher income per capita may increase public health expenditure, spending on female education and the number of physicians per capita. Subbarao and Raney (1993) are comparing the partial effect of income (the effect not operating through the other variables in their model) to the full effect of increased female education. The entire income effect, taking into account its impact through female education and the number of physicians, may be greater than the impact of female education on infant mortality. A corollary is that increasing female education may not be the most effective way to reduce infant mortality.

Singh (1994) examines the relationship between women's education and infant mortality. This paper argues that educating women increases the economic value of their time, raising the cost of having children. This in turn leads to decreased fertility and infant mortality. This paper formally models this relationship by regressing fertility and infant mortality, in two separate models, against measures of female education and economic worth. This paper concludes that the percentage of births attended by a trained health personnel, the percentage of women participating in the labour force, the percentage of women attending school and GNP per capita all affect infant mortality. The paper concludes that increased participation of women in schooling and the labour market raises the economic value of their time and thus the cost of having children. This in turn leads to lower fertility and infant mortality.

We can make three broad criticisms of this study. First, the general applicability of this model can be called into question as some of the regressions contain data from just 27

countries. Second, this paper uses a linear functional form despite the evidence discussed earlier that the relationship between per capita GDP and infant mortality is non-linear. And third, while this model provides evidence that women's education and labour market participation determine infant mortality, no specific evidence is provided that they do so through fertility.

Schell et al (2007) examine cross-country rates of infant mortality from 152 countries using WDI data. Their econometric results show that GNI per capita, income inequality and female illiteracy are significant determinants of mortality in low, middle and upper-income countries, but that public spending on health and poverty rates have no impact on infant mortality. This paper does not specify the causal chain through which female illiteracy affects child death rates.

In summary, cross-country multivariate studies provide substantial evidence that female education affects infant and child mortality. The different causal pathways through which female education could affect infant mortality are further discussed in the next chapter.

General Education and Male Education

Nine of the econometric studies we reviewed found that either education in general or male education reduce child mortality. This section discusses studies by Younger (2001) and Jamison et al (2006), which find that education, or male education, are significant determinants of child mortality.

Younger (2001) applies a growth regression approach to identifying the cross-country determinants of infant mortality. This paper formally tests whether infant mortality rates are converging over time before examining the determinants of infant mortality.

This paper tests for absolute convergence using country level data from the Global Development Network. Infant mortality rates display absolute convergence but at a very slow rate. By absolute convergence the paper means that when policies and structural factors are not controlled for, countries with a higher initial infant mortality rate see a larger absolute decline in their infant mortality rate than countries with a lower initial infant mortality rate. This means that over time countries with higher initial infant mortality rates are converging towards countries with lower rates of infant mortality.

Younger (2001) also examines policy variables that affect the rate at which infant mortality declines. This paper concludes that only two policy variables, namely primary school enrolments and DPT vaccinations for infants, are consistent determinants of infant mortality. This paper also concludes that there is little evidence that the availability of healthcare (as measured by doctors, nurses and hospital beds per 1000 of the population) determines infant mortality and that there is only limited evidence that GDP per capita is associated with infant mortality.

This paper's conclusion that few variables robustly affect infant mortality is drawn from models that include country fixed effects. This is equivalent to including a dummy variable for each country. Systematic variation in the rate of infant mortality between two countries will be accounted for by these dummy variables; this increases the explanatory power of the model in a purely statistical sense but it does not tell us why countries have consistently different rates of infant mortality. The systematically higher infant mortality in a country

must be due to some relevant independent variable or variables and arguably these and not dummy variables should be included in the model. The aim of a multivariate study is to explain variation in the dependent variable by including relevant independent variables and not just to ascribe variation to dummy variables.

The paper also includes education and not female education as a dependent variable. This is surprising because most previous multivariate studies conclude that female education is a more likely determinant of infant mortality. Given that education and female education are highly correlated, general education could be acting as a proxy for female education in this model.

Jamison et al (2006) examine the relationship between education and infant mortality. This paper specifies a panel data regression covering 62 countries for ten-year intervals from 1960 to 2000. When controlling for country fixed effects, they find that time, education quality, logged GDP per capita, whether a country is tropical or not, and technological progress are significant determinants of infant mortality. International student achievement tests in mathematics and science are used to measure education quality.

This paper concludes that there is a strong association between education quality and declines in infant mortality; with education having as strong an impact on infant mortality as income. Their main policy conclusion is that governments should improve education to reduce infant mortality.

Jamison et al (2006) provide strong evidence that education quality affects infant mortality. Yet they do not provide a strong theoretical argument for including education as opposed to female education and nor do they empirically test the impact of the quality of female education. Education and female education may be highly correlated. This means that education may be acting as a proxy for female education in their model. Thus the principal conclusion that we can draw from this paper is that it is the quality, and not just the amount, of education that determines child health: not that it is general education as opposed to female education that determines child health.

This section has reviewed two studies that found that education reduced child mortality. Education and women's education are likely to be strongly correlated across countries. The models reviewed in this section did not consider this and did not test for the significance of women's education. This means that education in these models could be acting as a proxy for women's education. There are theoretical reasons for considering that women's education will have a larger impact on child mortality than education. For female education could reduce child mortality by reducing fertility, increasing the resources children consume, improving access to healthcare and improving knowledge of health interventions. In contrast general education would mainly affect mortality by increasing knowledge of health interventions. So overall the evidence demonstrates that female education is more likely to be a significant determinant of child mortality than education.

Healthcare

Some multivariate studies argue that health interventions reduce child mortality. Others examine the impact of public health spending on child mortality. A few multivariate studies examine the impact of state capacity on child mortality. We discuss studies which examine

each of these determinants in more detail below. We include state capacity in this healthcare section as most multivariate studies argue that it reduces child mortality by increasing the effectiveness of public health spending.

Health Interventions

There is strong evidence from non-multivariate studies that health interventions (such as child vaccinations and the number of trained health professionals) reduce child mortality. Bryce et al (2003) for instance demonstrate that in 42 countries where 90% of child deaths occur, 63% of these deaths could have been prevented by the full implementation of a few well known and effective interventions. Multivariate studies also show that health interventions reduce infant and child mortality; with nine of the studies we reviewed finding this variable significant. However, only one paper, Anand and Baernighausen (2004), concentrates on health outputs' impact on child mortality.

Anand and Baernighausen (2004) examine the impact of human resources on child mortality. This paper argues that many worldwide child deaths could be averted by treatments provided by health professionals. The model specified in this paper shows that income (as measured by GDP per capita in PPP), poverty (as measured by the proportion of the population living on less than \$1 a day), female literacy and human resources for health are all significant determinants of infant and under-five mortality. Human resources are measured through a combined index showing the number of nurses, physicians or midwives per capita. When nurses and physicians are included separately, physicians are found to be significant but nurses are not. The authors conclude that investing in human resources for health should be explicitly considered as part of a strategy for achieving the MDGs.

We can make three main criticisms of this paper. First, the theoretical case for the medical interventions listed requiring a health professional to administer is debateable. Many of these medical interventions such as oral rehydration therapy and vaccines can, and are, administered by people with more limited medical training. Second, these authors do not include other outputs from health expenditure in their regression. However, countries that have more doctors are also likely to spend more on other health outputs such as hospitals, health centres, drugs etc; so human resources for healthcare may simply be acting as a proxy for overall health outputs or spending. This means that it may be more effective for developing countries to increase these other health outputs. Third, it is incorrect to assume that simply because human resources are a significant determinant of child mortality, spending more on health professionals in isolation would be cost effective. The effectiveness of physicians in reducing child mortality is probably dependent on a reasonable amount being spent on health equipment and facilities.

This section discussed one study which examined the impact of health outputs on mortality. The results of this study may be questioned because of the methodological and theoretical criticisms outlined above. There is, nevertheless, a strong theoretical case and much non-multivariate evidence for health outputs – such as vaccinations – affecting mortality. This evidence coupled with the nine econometric studies that find health outputs significant suggests that this independent variable may well explain some of the variation in child mortality rates across countries and time.

Public Expenditure on Health

There is much debate concerning the effectiveness of government health expenditure in reducing child mortality. Four sources of empirical evidence strongly suggest that government health spending can effectively reduce mortality. First, epidemiological studies (those that look at the direct/medical causes of death) note that the majority of child deaths in developing countries occur from a few well-understood and medically preventable diseases (Black et al 2003). Second, studies that analyze health interventions conclude the well known, proven, cost effective public health interventions are available for the majority of diseases that children die from worldwide (See Jones et al 2003). Third, cross-country multivariate studies find that health outputs, such as doctors per 1000 of the population and immunization rates, reduce mortality (Anand and Baernighausen (2004) and Wang 2002). And fourth, case studies, such as Victora et al (1996) and Gutierrez (1994), show that some governments introduced programmes which effectively reduced child mortality.

Yet multivariate studies that directly estimate the impact of government health expenditure on mortality appear to contradict these results. Filmer and Pritchett (1999) undertake one relatively recent study which examines the relationship between public health spending and infant and under-five mortality. Their panel data econometric model shows that per capita GDP, public spending on health as a fraction of GDP, female education, whether a country is Muslim or not and ethnic linguistic fractionalization are statistically significant determinants of infant and under-five mortality. This paper tests the robustness of these results by dropping outlying country observations, using the median regression technique and using data from two different datasets (UNICEF and WB). These techniques confirm that their results are robust.

Filmer and Pritchett (1999) argue that while public health expenditure is statistically significant its actual impact on mortality is small. Indeed according to their model even if countries doubled their public spending on health as a percent of GDP this would only lead to a four percent reduction in under-five mortality. This small impact translates into extremely high cost; with the cost of averting a child death in a typical developing country through increased public expenditure on health being over \$50,000.

We can gain a greater understanding of this apparent paradox by further examining the exact methodologies used by epidemiological and health intervention studies. Epidemiological studies, such as Black et al (2003), start by examining the number of children that die from a particular disease. These studies find that most children die from curable, well understood causes of death. Health intervention studies build on these epidemiological studies by showing that proven, cost effective health interventions exist for most of these causes of child deaths and thus health spending could rapidly reduce mortality (Jones et al 2003). Other studies have concluded that public health interventions are potentially cost effective. (Mosley and Jamison 1991 and Bryce et al 2003).

The problem with this methodology, from the viewpoint of examining health expenditures actual effectiveness, is that it implicitly assumes that all additional public health expenditure will be aimed at efficiently reducing mortality. Yet the unfortunate reality is that some developing countries' governments will use additional expenditure for the purpose of political patronage or prestige tertiary health expenditures (See Castro-Leal et al 2003 and Victora et al 2003). Many developing countries may also lack the capacity to rapidly scale up health

interventions (Schell et al 2007). Health intervention studies show the potential effectiveness of government expenditure across a range of interventions.

Studies of government expenditure in particular countries, meanwhile, show that specific countries have effectively reduced infant and child mortality (Alailama and Sanderatne 1997, Krishnan 1997, Victora et al 1996 and Gutierrez 1994). Yet this does not mean that health expenditure across all countries has been effective. Cross-country regressions measure the average effectiveness of government expenditure across all nations. This can lead to the conclusion that health expenditure cannot reduce infant or child mortality - an erroneous conclusion based on the methodological error of confusing the existing average relationship with the potential relationship. The regression has shown that on average across all countries expenditure has not reduced mortality: not that government spending cannot potentially reduce infant mortality. So, in summary, the evidence from econometric and health intervention studies shows that government health expenditure can potentially improve child survival, but that on average across countries government health expenditures' actual impact has been low. There may be a subset of countries with high capacity governments and strong governance where public health spending effectively reduces child mortality.

Governance and State Capacity

This section discusses studies by Lazarova (2006), Lena and London (1993), Navia and Zwiefel (2003) and Rajkumar and Swaroop (2007) which examine the impact of politics or state capacity on child mortality.

Lazarova (2006) examines the impact of governance on infant mortality across countries. Governance is measured by averaging four WB CPIA indicators, namely government effectiveness, regulatory quality, rule of law and control of corruption. This paper concludes that governance, GDP and income inequality are significant determinants of infant mortality across countries. And that governance explains more of the cross-country variation in infant mortality than income inequality.

One criticism of this study is that it uses a narrow definition of governance. The WB indicators used are arguably all measures of the capacity of government institutions. Government capacity may well affect the efficacy of public spending, but so may its political ideology. Left-wing governments may, for instance, have superior policies to right-wing governments for reducing child mortality.

Lena and London (1993) examine the impact of economic and political variables on under-five mortality. This paper uses a lagged cross-sectional analysis, whereby cross-country variation in the dependent variable at one point in time is explained by independent variables at an earlier point in time. The regression demonstrates that strong left-wing regimes decrease, and strong right-wing regimes increase, under-five mortality. But weak regimes of both political ideologies do not affect child health. The regression also shows that democracy and GNP per capita reduce, and investment dependency increases, under-five mortality. This paper concludes that strong left-wing regimes reduce under-five mortality.

This paper can be criticized for two reasons. First it uses central government expenditure as a percentage of GNP to measure state capacity/strength. However, this is clearly a measure of the size of government expenditure and not a government's capacity. Some governments

make substantial expenditures but have poorly trained civil servants, inefficient bureaucracies and high levels of corruption; such states do not have high capacity (Kohli 2004). It could also be argued that the complex politics of developing countries cannot be adequately captured through a leftist, centralist and rightist classification. Some governments in developing countries may, for instance, proclaim that they are following progressive policies to justify government spending which mainly benefits a small elite (Kohli 2004 and Victora et al 2003).

Navia and Zweifel (2003) investigate the relationship between a country's politics and infant mortality. They use the Heckman Two Step multivariate method to isolate the impact of a country's political regime on infant mortality. They conclude that, controlling for other factors, democracy lowers and dictatorship increases infant mortality. Their model also shows that fertility increases, and labour force participation by women decreases, infant mortality. Immunization rates and Official Development Assistance also decrease infant mortality, in this model, but only in democracies.

This paper also attempts to establish the mechanism through which democracy reduces infant mortality. It argues that democracies are associated with higher social spending, which reduces infant mortality. Yet this argument is unconvincing because this paper fails to provide any empirical evidence that democracies spend more or that higher spending reduces infant mortality. And as discussed earlier, most multivariate studies conclude that public expenditure has a limited impact on infant mortality.

We can also question the basis through which this study categorizes countries. A country is categorized as a democracy if there are regular elections and changes of government. Commentators such as Schmitter and Karl (1991) have, however, argued that there is more to a democracy than just having elections, and that a free press, free speech and a universal franchise are intrinsic characteristics of, and not optional extras for, democratic nations. There are many developing countries such as Nigeria which claim to be democratic and have regular elections but where such elections are marred by violence, intimidation, the stuffing of ballot boxes and vote buying. It is debatable whether such countries can be easily classified as either democratic or a dictatorship.

The studies by Lazarova (2006), Navia and Zweifel (2003) and Lena and London (1993) provide some evidence that governance, political ideology and democracy may affect child mortality across countries. These papers do not, however, directly show that these variables make public health spending more or less effective. Countries with good governance may have more effective public health spending. But it could also be the case that such governments have more effective regulatory environments for private health spending or make more effective expenditures on other relevant social policies. These studies do not therefore explain why public health spending can potentially, but on average across countries actually does not, reduce child mortality.

Rajkumar and Swaroop (2007) directly model how the effectiveness of health spending in reducing child mortality varies with countries' governance. This paper finds that in countries with good governance public health spending is more effective in reducing child mortality than in countries with average governance. And that in countries with poor governance public spending has no impact. This study is a useful attempt to measure the impact of governance on the efficacy of public health spending. We can, however, criticize this study because its measure of governance is based on a corruption index which does not account for the

education level of the civil service, the effectiveness of government institutions or the impact of politics on the efficacy of public expenditure.

In conclusion, there is compelling evidence that public health spending could cost effectively reduce child mortality, but that its average actual impact across countries has been low. There may, however, be a subset of countries with high capacity governments and amenable governance where public health spending has effectively reduced child mortality. Studies which have further examined this issue have both narrowly defined state capacity and governance or have not directly shown that these variables affect child mortality through public health spending. This issue thus warrants further empirical analysis.

Environmental Factors

Environmental factors may affect the spread of diseases and child mortality. Epidemiological studies emphasize that water supply and sanitation affect the spread of many infectious diseases from which significant numbers of children die (Esrey et al 1991). This section discusses multivariate studies by Fay et al (2003) and Franz and Fitzroy (2006) which find that environmental factors are significant determinants of child mortality.

Fay et al (2003) investigate the causes of infant mortality using a database of mortality rates by quintiles of wealth for 43 developing countries from DHS. The regressions carried out in this paper show that GDP per capita, ethno-linguistic fractionalization, the Gini index, female illiteracy rate, underweight at birth, lower mother body mass index, access to piped water, and the four asset quintiles were significant determinants of infant mortality. This paper concludes that in addition to income; assets, female education, direct health interventions and better access to piped water have an important role to play in improving child health outcomes.

Franz and Fitzroy (2006) examine the determinants of child mortality in 61 developing countries. This paper found that calories available per day, mean consumption of the poor, female participation rate in agriculture, female literacy, incidence of tuberculosis, estimated total fertility rate and regional dummies were significant determinants of child health. This paper concludes that the significance of the regional dummy for the Central Asian Republics (CAR) shows that excess mortality occurs in this region and it goes on to argue that previous medical and epidemiological evidence shows that environmental degradation caused this excess mortality. The conclusion reached by Franz and Fitzroy can, however, be criticized because there are many possible explanations for CAR countries dummy variables being significant. It may, for example, be the case that this dummy variable is picking up the impact of poor governance or the sharp decline in the quality of healthcare that occurred after these countries became independent and stopped receiving inter-governmental transfers from the USSR.

In summary, there is a strong theoretical case that improved clean water and sanitation reduce disease prevalence and child mortality. Yet there is only limited empirical evidence for this theory from the multivariate studies reviewed here. The most likely explanation for this is that the variables that many multivariate studies find significant partially affect child mortality through improved water supply and sanitation. Higher GDP per capita, for example, may reduce child mortality through better water supply and sanitation. A regression that included both socioeconomic variables and water supply and sanitation would find water

and sanitation significant because they are the more direct determinant of child mortality. However, most multivariate studies have not included water supply and sanitation but have included socioeconomic determinants that may work through these variables. There is a strong argument for considering that water supply and sanitation partly determine child mortality. And that the main reason that most econometric studies reviewed in this chapter did not find these variables significant is simply because they failed to test for them.

Proximate Determinants

There is overwhelming evidence from studies that examine child mortality across families in particular countries that biological and demographic factors such as birth spacing and mother's age at conception affect infant and child mortality (Miller et al 1992 and Forste 1994). Yet only five¹⁵ of the studies reviewed in this chapter find that these or other proximate variables significantly affect child mortality. The reason for this apparent paradox is that most cross-country studies use country level data and deliberately examine socioeconomic and not proximate causes of child deaths.

This section discusses three studies (Hobcraft et al (1985), Heaton et al (2005) and Rutstein (2000)) that examine the proximate determinants of mortality. The other three studies included in our detailed literature review which found proximate variables significant are not examined here because they concentrated their discussion on other socioeconomic variables and only included proximate variables in an attempt to properly specify their regression equations.

Hobcraft et al (1985) examine the determinants of infant and early child mortality. They do so using data on individuals/families from 39 World Health Surveys. Using individual level data for infant and child deaths, the authors calculate a separate regression equation for each country. Their results show that birth order, birth spacing, proximity to subsequent births, a mother's education and children being born to teenage mothers are significant determinants of infant, neonatal, post-neonatal, under-two and two to four year mortality. This paper emphasizes that teenage mothers and birth spacing are the two most important determinants of infant and early child mortality.

This study includes female education and birth spacing as independent variables. Female education may, however increase the value placed on women's time, reducing fertility and increasing birth spacing. It follows that this regression only shows the partial effect of mother's education and may underestimate its impact on infant and early child mortality.

Heaton et al (2005) evaluate the determinants of infant mortality across families and countries. This study pools family level data from 42 countries' DHS, giving 160,000 observations. This paper finds that income, mother's education and breastfeeding significantly reduce infant mortality in many different countries. These authors conclude that programmes to improve female education and encourage breastfeeding could substantially reduce mortality.

¹⁵ See: Hobcraft et al (1985); Rutstein (2000); Fay et al (2003); Heaton et al (2005) and Franz and Fitzroy (2006).

Rutstein (2000) examines the determinants of infant mortality in the 1990s. The dependent variable in this study is the percentage change in infant mortality between two DHS for each country's rural and urban areas, so there are 33 countries and 66 observations. This study concludes that four sets of variables, namely biological (birth order, age of mother, underweight, wasting), medical access (prenatal care, birth attendance, measles vaccination, diarrhoea treatment, oral rehydration), environmental (water supply, toilet facilities, dirt flooring) and socioeconomic (electricity, education), determine mortality. The paper concludes that we cannot attribute changes in infant mortality to a few factors. Rather a broad set of factors are associated with infant mortality and countries that did substantially improve child health did so by improving a host of factors.

The studies by Heaton et al (2005) and Hobcraft et al (1985) provide strong evidence from large samples of family level data that proximate variables partially determine infant mortality. Rutstein (2000) also provides some evidence that these variables affect variation in infant mortality across countries. There is also strong evidence from econometric studies, which examine family level data in specific countries, that proximate variables affect mortality. The most important proximate variables are probably birth spacing and mother's age at birth.

Linkages between the Multivariate Studies Reviewed and our Econometric Model

This section discusses the functional form, lag structure, type of econometric model and tests for robustness used by the multivariate models we have reviewed. We also discuss how these influence the econometric model we specify in chapter 4.

Functional Form

Filmer and Pritchett (1999) postulate that there is a strong theoretical argument that the relationship between child mortality and income will be non-linear. Infant, under-three and under-five mortality are all bounded at zero while GDP per capita does not have an upper-bound. This implies that as income increases it must reduce child mortality by a diminishing amount. Epidemiological and medical evidence also support this argument. In countries with high under-five mortality many child deaths occur from infectious diseases for which cost effective medical interventions exist which could sharply reduce child mortality. Relatively small increases in expenditure on a few cost effective health interventions could sharply reduce under-five mortality in such countries. In contrast, in richer countries where child mortality rates are lower more children die from non-infectious diseases which are more costly to treat. In addition, a small increase in household income and consumption in poor households could sharply reduce under-nutrition, making children less prone to morbidity and mortality from many diseases (Mosley and Chen 1984). In richer countries under-nutrition is no longer a problem and it could even be argued that too higher family income has led to over-consumption, obesity and related health problems. There is much evidence to suggest a non-linear relationship between income and child mortality across countries.

This argument is recognized in the literature and most multivariate studies specify non-linear functional forms; with some including a log of income (Younger 2001) and others using a quadratic functional form (Rodgers 1979). Our econometric model follows the literature in

specifying a non-linear relationship between income and child mortality with many of our models including a log of income.

Many multivariate studies such as Pritchett and Summers (1996) use a double log functional form. This has the convenience of easy interpretation of the coefficients which can now be read as showing the elasticity between income and child mortality. The imposition of a constant elasticity may, however, be incorrect because as child mortality approaches its lower bound, an ever larger percentage increase in income could be required to achieve the same percentage change in child mortality. Pritchett and Summers (1996), however, argue that in samples of developing countries this is a moot point as child mortality levels are not approaching the very low levels where the relationship is highly non-linear. We argue that whether there is or is not a constant elasticity between income and child mortality in our sample is an empirical question. Thus in the econometric analysis in part three we divide our sample into numerous income groups and examine whether the elasticity between income and child mortality consistently and significantly decreases as we move from low-income to upper-middle-income countries.

Lag Structure

Some of the multivariate studies discussed in this paper argue that the independent variables should affect child mortality with a five year lag (Shen and Williamson 1999; while others (Jamison et al 2006, and McGuire 2006) do not specify any lag. We discuss the lag structure for three variables (income, income inequality and women's education) for which there is substantial evidence.

Income at the national level could affect child mortality with a lag through increased government spending leading to better healthcare. Alternatively, income at the national level could immediately be associated with lower child mortality through higher family income reducing mortality due to higher consumption. Evidence from Filmer and Pritchett (1999) suggests income at the national level is mainly associated with child mortality through family income and therefore most of our models do not put a lag on this variable.

The power of women could affect child mortality with a lag if it represents their ability to influence government policy and the distribution of public health expenditure. Yet there is also a strong argument that women's power could immediately reduce child mortality if it is working through a mother's control over household resources. Most studies have stressed mothers' control over household resources and not their influence on policy making (Caldwell 1986 and Hobcraft et al 1985) and therefore we model this relationship without a lag in chapter 4.

Income inequality is also modelled without a lag in our econometric model. The reason for this is that we follow Rodgers (1979) in arguing that there is a relationship between income inequality and child mortality at the national level because there are diminishing marginal returns between family income and child mortality at the individual level.

Type of Econometric Model (Cross-Country or Panel)

The multivariate models discussed in this chapter mainly use either cross-country datasets or panel datasets where the units are different countries at different points in time. Most of the earlier studies such as Rodgers (1979) and Flegg (1982) use cross-country datasets. More

recent studies such as Pritchett and Summers (1996), Asafu-Adjaye (2004) and Rajikumar and Swaroop (2007) use panel datasets.

There are both advantages and disadvantages to using panel data. The advantages are that it increases the size of the dataset available, provides more information on the variables and allows us to model changes over time. One disadvantage to using panel data is that some observations may change slowly over time and be highly correlated with their previous values. This means that although panel data often gives additional observations it sometimes provides limited additional information. The use of panel data often complicates the error structure and means stationarity has to be tested for and dealt with.

We deal with this problem in chapter 4 by first estimating a cross-country model and then estimating a time fixed effects panel data model. First estimating a cross-country model allows us to test our hypotheses without worrying about some complex statistical issues such as stationarity and auto-correlation. The introduction of a panel data model allows us to see whether our independent variables can also explain changes in child mortality over time and increases our sample size.

Some previous models such as Asafu-Adjaye (2004) use panel data models but do not test for stationarity. This is problematic because if the independent variable is non-stationary (is increasing or decreasing in the long term) but the dependent variable is stationary (moving around a constant mean) then clearly there cannot be a long term relationship between them even if the coefficient is statistically significant. Likewise if both variables are non-stationary the relationship between them could be occurring because they are both following a similar trend over time (spurious regression). The only exception would be if short term changes in the independent variable caused short term changes in the dependent variable (cointegration). In chapter 4 we test all the independent and dependent variables for stationarity and formulate and interpret our model in light of these results.

We also build on earlier work when selecting the exact type of panel data model. Pritchett and Summers (1996) estimate their model using country and time fixed effects. We do not use country fixed effects in our model because the dummy variable for each country in such models absorbs the systematic variation in child mortality between countries. We argue that this is problematic because we are trying to explain why some countries have systematically higher child mortality than others by including relevant explanatory variables and not simply attributing such variation to dummy variables. Our panel data econometric model does, however, include time fixed effects to account for advances in health technology which are driven by research spending in OECD countries which are not included in our dataset.

Interaction Terms

Rajikumar and Swaroop (2007) argue that governance, public spending and health outcomes may be linked. Their model includes under-five mortality as a dependent variable and includes governance (quality of bureaucracy and corruption) interacted with public health spending as a percentage of GDP as an independent variable. Their results show that public spending is more effective in reducing under-five mortality in the presence of good governance. The argument that governance or state capacity may partially determine the efficacy of public health spending seems intuitively plausible. We build on this work by including interaction terms between public health spending and measures of state capacity and governance. We also specify models which include interaction terms between total health

spending and good governance; our argument being that state capacity, through regulation of the health industry, may influence the efficacy of private health spending.

Test for Robustness

Pritchett and Summers (1996) test their results for robustness by using two alternative measures of child health, namely infant mortality and under-five mortality. Their argument is that because these two measures are closely related, a model which predicts one should also explain the other. Our econometric model uses a similar test for robustness by using the following different measures of child survival: neonatal, post-neonatal, infant, under-three and under-five mortality as dependent variables. We also test whether our model can explain differences in child nutrition as this may be an effective measure of a child health status and be closely related to child survival.

Pritchett and Summers (1996) also argue that using different datasets can provide an appropriate test for the robustness of econometric results. Similarly we test our econometric model using WDI and DHS data. We also construct our own dataset of under-three and infant mortality based on family level data from DHS and include these data as dependent variables in our model to test for robustness. The technique we use to construct this dataset is discussed later on in this thesis.

Conclusion

This section has discussed how the multivariate studies reviewed influenced the lag structure, functional form and robustness tests we use when specifying our econometric model in chapter 4. It also discussed how recent papers have used panel data models and interaction terms and how our model builds on these econometric techniques.

Criticisms and Conclusion

This chapter has discussed existing empirical evidence on the determinants of child mortality. We can, however, criticize many of these multivariate studies because they do not specify a detailed analytical framework and because they may use inaccurate data. These two problems are further discussed below.

The most serious problem with many earlier econometric studies is that they do not specify an analytical framework. That is they do not explicitly state the causal pathways through which the independent variables affect child mortality and interact with each other. Yet without such a framework it is difficult to derive meaningful policies to reduce child mortality and achieve MDG4. For example, studies by Hanmer et al (2003) and Easterly (1999) argue that national income improves child health but fail to identify the causal mechanism through which this occurs. We cannot, however, derive meaningful policies to achieve MDG4 unless we know how income reduces child mortality. For if national income reduces child mortality by providing increased resources for social expenditure then the obvious policy conclusion is that more social spending is needed to achieve MDG4. But if national income is reducing mortality solely through its association with increased private income then the policy conclusion that more social expenditure is needed to achieve MDG4 is erroneous.

Other multivariate studies discuss, but do not formally specify or test, how significant independent variables affect child health. Flegg's (1982) study, for instance, shows that female literacy reduces child mortality but does not formally test whether it does so by increasing women's power, their economic worth (leading to reduced fertility) or mothers' knowledge of childcare. Yet the causal mechanism between women's education and mortality affects the policies a government should implement. If the "knowledge of childcare" mechanism is correct then governments should concentrate on specific/ targeted education. If the "women's power" mechanism is correct then governments should concentrate on improving women's status within society.

The lack of an analytical framework defining the causal chains between different variables also makes it difficult to properly interpret the coefficients on the independent variables in many studies. Subbarao and Raney (1993), for instance, show that female secondary school enrolment, GDP per capita and population per physician are all significant determinants of child mortality. Using the coefficients on these variables they show that doubling female secondary school enrolment would reduce mortality by a much greater amount than doubling GDP. The authors conclude that increasing female secondary school enrolment should be prioritized over increasing income. Yet this may be an incorrect interpretation of the model's results. Higher national income could lead through higher social spending to more physicians, teachers, schools and higher female enrolment; meaning the coefficient on income in their model would show its partial effect. So this paper may have compared the partial impact of income to the full effect of female education - an unfair comparison. The entire income effect, taking into account its impact through female education and the number of physicians, may be greater than the impact of female education on child mortality. A corollary is that increasing female education may not be the most effective way to improve child health.

The policy conclusions identified by many multivariate studies are also overly general. For instance, Anand and Baernighausen (2004) find that income, poverty, female literacy and human resources for health are all significant determinants of infant mortality. The authors conclude that investing in human resources for health should be explicitly considered as part of a strategy for achieving MDG4. Yet only a foolish government would start employing more doctors based on the general result that health professionals explain some of the variation in infant and child mortality across countries. Deriving realistic policy recommendations involves more than identifying simple statistical relationships between variables across countries.

The lack of an analytical framework can also make it difficult to test the robustness of different models. For instance Hanmer et al (2003) run hundreds of regressions with different combinations of independent variables and infant and two to four mortality as the dependent variable. They rate the robustness of different independent variables according to the number of regressions in which they were significant. According to their results the most robust determinants of mortality are male primary school enrolments and GNP per capita. This paper also concludes that some health outputs such as measles vaccinations are robust determinants of two to four mortality. This paper's conclusions may be problematic for two reasons. First, for econometric results to be meaningful there must be a strong theoretical argument for a particular independent variable affecting the dependent variable. Yet Hamner et al (2003) fail to explain how male primary school enrolments would reduce mortality and previous empirical and theoretical work provides compelling evidence that female education

should have a stronger impact on child mortality. Second, the causal relationship between different independent variables affects their chances of being significant. The finding that health professionals do not affect mortality, for instance, could be because their impact occurs through other variables – such as vaccination rates – which are included in the model. The regression technique used by Hanmer et al (2003) has then an intrinsic bias towards choosing variables that directly affect infant and child mortality compared to socioeconomic variables that operate through other more proximate variables.

Many multivariate studies can also be criticized for using inaccurate data. The vast majority¹⁶ of studies use data from international datasets. These datasets contain, because the academic and donor communities demand, data which are comprehensive in that they cover nearly all countries for many years. Yet most developing countries lack the vital registration systems necessary to collate yearly under-five mortality data. The cross-country databases¹⁷ provided by international organizations are therefore based on data collected via vital registration systems, national censuses, multiple indicator clusters and DHS. This leads to two problems. First, different sources give different estimates of under-five mortality, suggesting that data from different sources may not be strictly comparable across countries or periods. Second, for some countries and years actual data does not exist, so the reported under-five mortality rate is itself predicted based on past trends (Murray et al 2007). Analysis based on such data is likely to predict trends which are determined by the rules and assumptions governing the original interpolations and not the true rate of decline or increase in infant or child mortality data (Bhargava et al 2001).

In conclusion, multivariate studies provide considerable evidence that income, income inequality, female education/power and health outputs determine child mortality across countries and time. Many multivariate studies find that general education affects child health but this is probably because it acts as a proxy for female education. There is also a strong theoretical argument, considerable evidence from epidemiological and health intervention studies and some evidence from multivariate studies that water supply and sanitation and proximate determinants affect mortality. Health intervention studies and case studies show that public spending on healthcare can potentially reduce mortality, but multivariate studies show that its actual average impact has been weak. Public health spending may, however, be more effective in reducing mortality in countries with amenable governance and high capacity governments. The next chapter discusses how each socioeconomic determinant for which there is considerable empirical evidence affects mortality through other more proximate variables. It also discusses the complex interrelationships between different socioeconomic variables.

¹⁶Only six studies used data from consistent surveys.

¹⁷ For many of the publicly available international databases such as World Development Indicators and the MDG database it was difficult to trace the original source of the data.

Chapter 3: Analytical Framework

Introduction

This chapter constructs an analytical framework for investigating the determinants of child survival in developing countries. The analytical framework models the causal pathways between different socioeconomic variables, between socioeconomic and proximate variables, and between proximate variables and child survival.

This chapter defines variables as either socioeconomic or proximate. The term socioeconomic is used to describe any variable that could affect child survival through a proximate variable. Income, income inequality and health spending are examples of socioeconomic determinants. A proximate determinant is any variable that can directly affect the chances of an infant or child contracting, or dying from, a medical cause of death. Birth order and a mother's age at birth are examples of proximate determinants. This framework is based on two main assumptions. First, that socioeconomic variables do not directly affect child survival: they operate through proximate determinants (Mosley and Chen 1984). And second, that some socioeconomic variables affect proximate variables through their impact on other socioeconomic variables.

Many multivariate regressions have investigated the determinants of child survival without explicitly using an analytical framework. Why, then, is an analytical framework necessary? There are two reasons. First, any model that specifies a relationship between a socioeconomic variable and child survival without specifying how it operates through proximate variables is essentially theory-less. Stating, as many multivariate studies do, that national income is associated with lower child mortality is insufficient: the mechanism through which income affects child mortality should be carefully investigated and explained. This is not just of academic interest: it has important policy implications. For example, if economic growth reduces child death rates by increasing private consumption, there is no need for social policy. Yet if economic growth reduces child death rates by providing resources for increased social expenditure, it is only because of the existence of social policies that child death rates have fallen. It follows that models that do not specify the causal pathways through which socioeconomic variables affect child health cannot be used to derive policy.

Second, without an analytical framework explicitly outlining the relationships between different socioeconomic and proximate variables, multivariate studies cannot correctly interpret the coefficients on their independent variables, and they may even erroneously conclude that a significant independent variable is insignificant (Victora et al 1997 and O'campo et al 1997). Say a cross-country regression finds that GDP per capita and vaccinations per 1000 children significantly increase child survival, but that public expenditure on health has no impact; without an analytical framework it is impossible to conclude whether: a) public expenditure is truly insignificant or b) that it can only reduce infant mortality by increasing vaccinations.

Mosley and Chen (1984) specified an earlier framework for the study of child survival. This chapter follows these authors in assuming that all socioeconomic variables must operate through proximate determinants. The classification of proximate determinants used in our

analytical framework also borrows heavily from this earlier work. Our analytical framework builds on this work by including and defining the causal pathways between actual socioeconomic variables for which there is substantial empirical evidence. This means we can use our analytical framework to formulate testable hypotheses.

The rest of this chapter is divided into four sections. Section one outlines the proximate determinants. Section two outlines the socioeconomic determinants. The third section interlinks the proximate and socioeconomic determinants. And the last section draws hypotheses which are empirically tested in chapter 4.

Analytical Framework of the Proximate Determinants

A proximate determinant is any variable that can either increase or decrease the chances of contracting, or dying from once contracted, a cause of death. A cause of death is any underlying cause of death recorded on medical certificates, such as malaria. Based on Mosley and Chen (1984), this proximate determinants framework assumes that:

- In the optimal setting at least 99%¹⁸ of infants will survive through the first five years of life. The higher rates of infant and under-five mortality seen in developing countries can, thus, be attributed to specific proximate determinants;
- That infant and under-five mortality are the cumulative consequences of many disease processes: only infrequently is a child's death the result of a single isolated disease episode;
- That growth faltering/under-nutrition is an accurate indicator of a child's overall health status, his/her susceptibility to death from a specific cause of death and the impact of all previous proximate determinants on the child's health.

The actual proximate determinants are divided into five groups. These are:

- Maternal factors (birth order, birth interval and a mother's age at birth);
- Environmental contamination (air, food, water, fingers, skin, soil, inanimate objects, insect vectors);
- Nutrient deficiency (calories, protein, micronutrients, vitamins, minerals);
- Injury (accidental and intentional);
- Personal illness control (personal preventative measures and medical treatment).

These proximate determinants are comprehensive. That is there are no other proximate determinants that affect child survival. And all other variables that affect child survival must either work through a proximate variable or work through a causal chain of variables that eventually affects a proximate variable.

¹⁸ Iceland has the lowest infant mortality rate of any country at 2.9 per 1000 live births (Source CIA World Fact Book April 2009).

Diagram 1 on the next page shows how these proximate determinants interact with each other and affect child health in an inter-temporal setting. In the first period, maternal factors, such as birth interval and a mother's age at birth, determine neonates' innate susceptibility to contracting a cause of death. Personal illness control then affects the chances that the neonate will contract a cause of death from nutrient deficiency, environmental contamination or injury. After exposure to contamination, illness, or nutrient deficiency, either the infant contracts a specific illness or injury that can lead to mortality, or it remains healthy in which case its health status relative to other infants is measured through growth faltering/under-nutrition. Infants that contract a cause of death die from it or recover, in which case it permanently affects their health status as demonstrated by growth faltering/under-nutrition. Ex-post illness control, such as medical treatments, can reduce that infant's chances of dying from a contracted cause.

Growth faltering/under-nutrition at the end of period one partially determines the chances of contracting and dying from a cause of death in period two. Maternal factors do not directly affect child mortality in the second period because their impact operates through the child's growth status at the beginning of the period. All the other proximate determinants affect child survival as they do in the first period.

This analytical framework uses growth faltering/under-nutrition as a measure of health status (Mosley and Chen 1984). A child's or infant's health status shows, holding the other values of the proximate determinants constant, the chances that the child will or will not contract, and will or will not die from, an illness or injury. Researchers have sometimes regarded growth faltering/under-nutrition as principally being due to dietary deficiency. This inference is probably incorrect; with most recent evidence suggesting that numerous factors cause growth faltering, making it an appropriate measure of overall health status¹⁹.

The proximate determinants analytical framework is simple and intuitive. Yet it also leads to the following important and empirically verifiable conclusions:

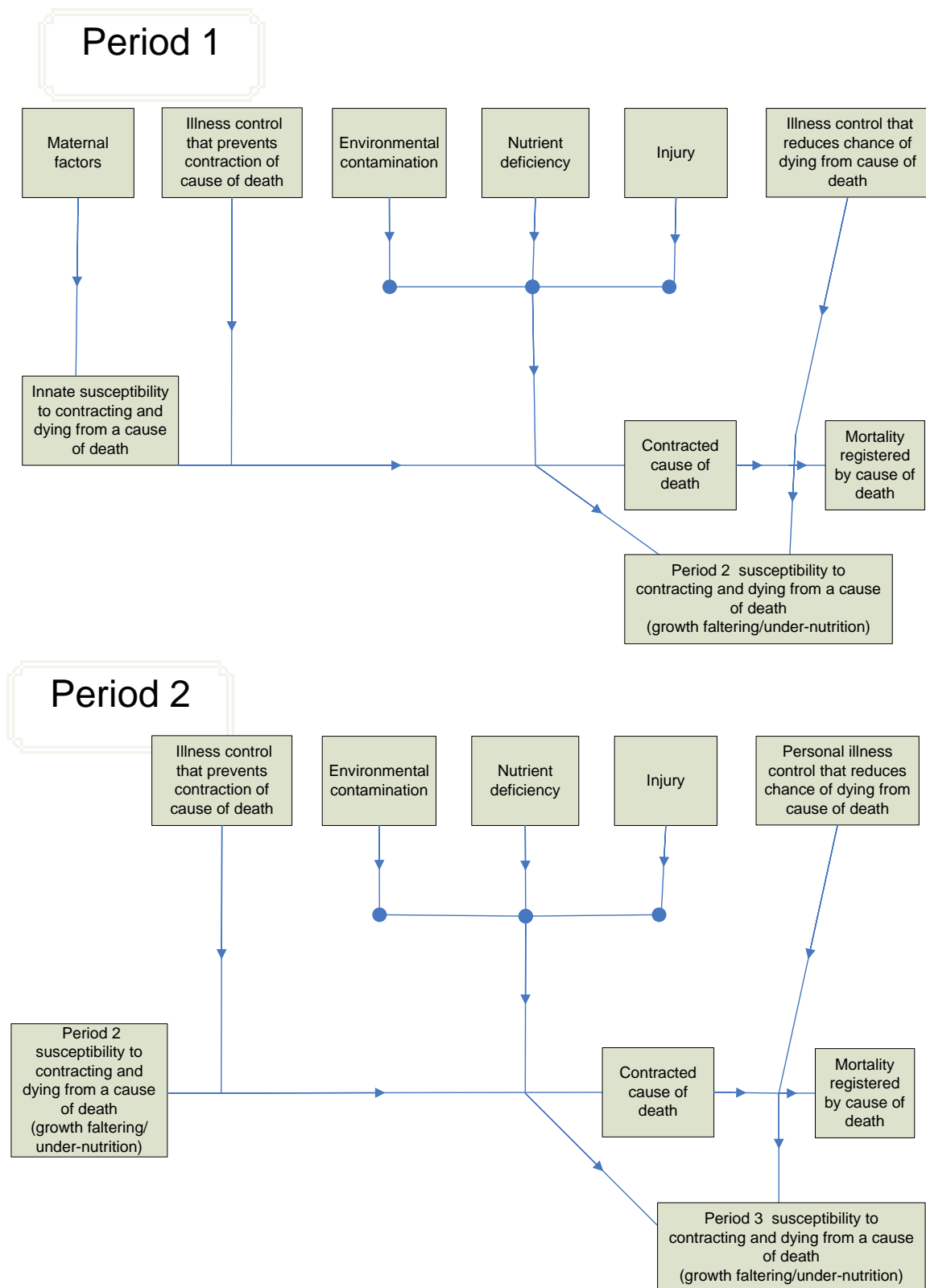
- All proximate determinants and socioeconomic determinants are potentially relevant in explaining mortality due to any specific cause of death;
- The determinants of child survival from all causes of death are also determinants of mortality from any single cause of death; if a) health status significantly affects the chances of contracting and dying from the single cause of death, and b) if ex-post health interventions are rare or ineffective;
- Over any period, proximate determinants can lead to diseases that cause growth faltering and/or child mortality. Over a short period child mortality may be a rare event. Studies should thus only examine child survival over long periods of time and large populations. Alternatively, the dependent variable should combine mortality and nutritional status²⁰;
- If we are seeking to explain child mortality in infant or children over a specific period, then growth faltering at the beginning of this period will be a significant explanatory factor;

¹⁹ This argument is taken from Mosley and Chen (1984). For evidence in support of this argument see (Mata 1978), Cole and Parkin (1977) Schroeder and Brown (1994) and Moore (2001).

²⁰ This conclusion is taken from Mosley and Chen (1984).

- Multivariate models that include both growth faltering and proximate variables as explanatory variables will only show proximate variables' partial impact. That is they will not show how proximate variables affect child mortality through growth faltering;
- A multivariate model that explains child survival should also explain the combined occurrence of child survival and growth faltering, as well as growth faltering in isolation;
- Different causes of death will lead to different rates of growth faltering and mortality. Some causes of death will have low mortality but high growth faltering; increasing the child's chances of dying from another cause of death in the future;
- It follows that there is no one to one correspondence between the number of deaths recorded as occurring due to a certain cause and the number of deaths it actually caused;
- Thus, studies of the cost effectiveness of different health interventions that assume that diseases that are recorded as a cause of death for X million deaths actually caused X million deaths will reach erroneous conclusions.

Diagram 1: Proximate Determinants of Child Health



Analytical Framework for the Socioeconomic Determinants of Infant and Child Mortality

This section creates an analytical framework showing the causal pathways between the different socioeconomic determinants of child survival. Creating such a framework involves making simplifying assumptions about how different socioeconomic variables interrelate. These assumptions are often controversial, and in truth, this chapter could have arranged the socioeconomic determinants of child survival in a myriad of ways, many of which would have been consistent with existing evidence. Some would regard this as an insurmountable problem. Yet all research makes simplifying assumptions about how society works. Most of the multivariate studies reviewed in the last chapter, for example, assumed that economic growth increases child survival, and not that improved healthcare both increases workers' economic productivity and reduces infant and child mortality. The argument is not whether research makes assumptions, but whether they are made explicitly or implicitly. By explicitly stating the envisaged causal pathways between socioeconomic variables, this chapter can formulate specific hypotheses to test these assumptions.

This chapter only briefly discusses the empirical evidence in support of different socioeconomic variables in the analytical framework. The reason is that this evidence has already been discussed in the literature review in chapter 2. To summarize, existing multivariate studies provide compelling evidence that income, income inequality, female education/power and health outputs affect mortality across countries and time. There is less, but still substantial, empirical evidence and a strong theoretical case that water supply and sanitation partly determine rates of child mortality. All these variables are, thus, included in the analytical framework.

Epidemiological, health intervention and programme/case studies show that public spending on healthcare can potentially, and in some countries has, improved rates of child survival. However, multivariate studies show that its actual average impact has been small. Some multivariate studies argue and provide empirical evidence that high state capacity is associated with more effective public health expenditure. State capacity may explain why public expenditure has been effective in some countries, but ineffective on average across all countries, in reducing child mortality. Our analytical framework includes, and shows the causal pathways between, state capacity, public spending and child survival.

Part 1 of this thesis discusses and uses the term state capacity. By state capacity we mean the ability of the government to formulate and implement social policies. Chapter 5, part 5 of this thesis discusses state power. By state power we mean the ability of the government to provide services to, cooperate with and control the population. Our concept of state power is then broader than that of state capacity.

Our analytical framework also hypothesizes that politics may affect the efficacy of social policy. We follow Heywood and Gamble (2003) in defining politics as the process through which different socioeconomic groups use their power to shape government policy. We label the power different socioeconomic groups have to determine government policy "distributional power". The inclusion of politics and the distribution of power between different socioeconomic groups as variables in our analytical framework can be criticized on the grounds that there is limited empirical evidence from existing multivariate studies that

these variables affect mortality. Yet the inclusion of these variables is justified because there is considerable direct and indirect evidence from other sources that these variables can affect the efficacy of social policy and state capacity. This evidence is discussed below.

There is evidence that poor children receive a lower quality of care than those in rich families in many developing countries (Victora et al 2003). Public health facilities in rich areas are often better funded by the government, have better trained staff and are better stocked with medicines (Lavy et al 1996). Few studies analyze the distributional power of those rich groups receiving superior services in detail, but it seems reasonable to assume that their ability to influence government policy may account for the superior and better funded services in the areas where they live. Shiffman (2007) analyzes the effectiveness and priority given to maternal mortality programmes in Guatemala, Honduras, Indonesia and Nigeria. He argues that the extent to which powerful political elites support maternal mortality programmes partly determines their effectiveness.

There is also much evidence from developed countries that the power of different socioeconomic groups to influence government policy determines the overall scope and effectiveness of social policies. Korpi (1983) argues that in Sweden an increase in the working classes' power to determine government policy led to the introduction of a comprehensive welfare state which improved welfare outcomes. Therborn (1986) found that business interests favoured the minimal private provision of healthcare, but that trade unions representing the working class favoured greater state intervention and universal health policies.

There is also evidence that societies which are fragmented develop weak states which cannot implement effective social policies. Alesina et al (2003) and Porta et al (1999) argue that in many ethnically diverse societies there is destructive competition and sometimes conflict between different ethnic groups, which contributes to a weak state which is unable to implement effective social policies. Kohli (2004) has argued that destructive competition between socioeconomic groups that are often formed along racial lines in Nigeria has led to a weak state and social policies which are principally a conduit for the distribution of resources to politically powerful groups.

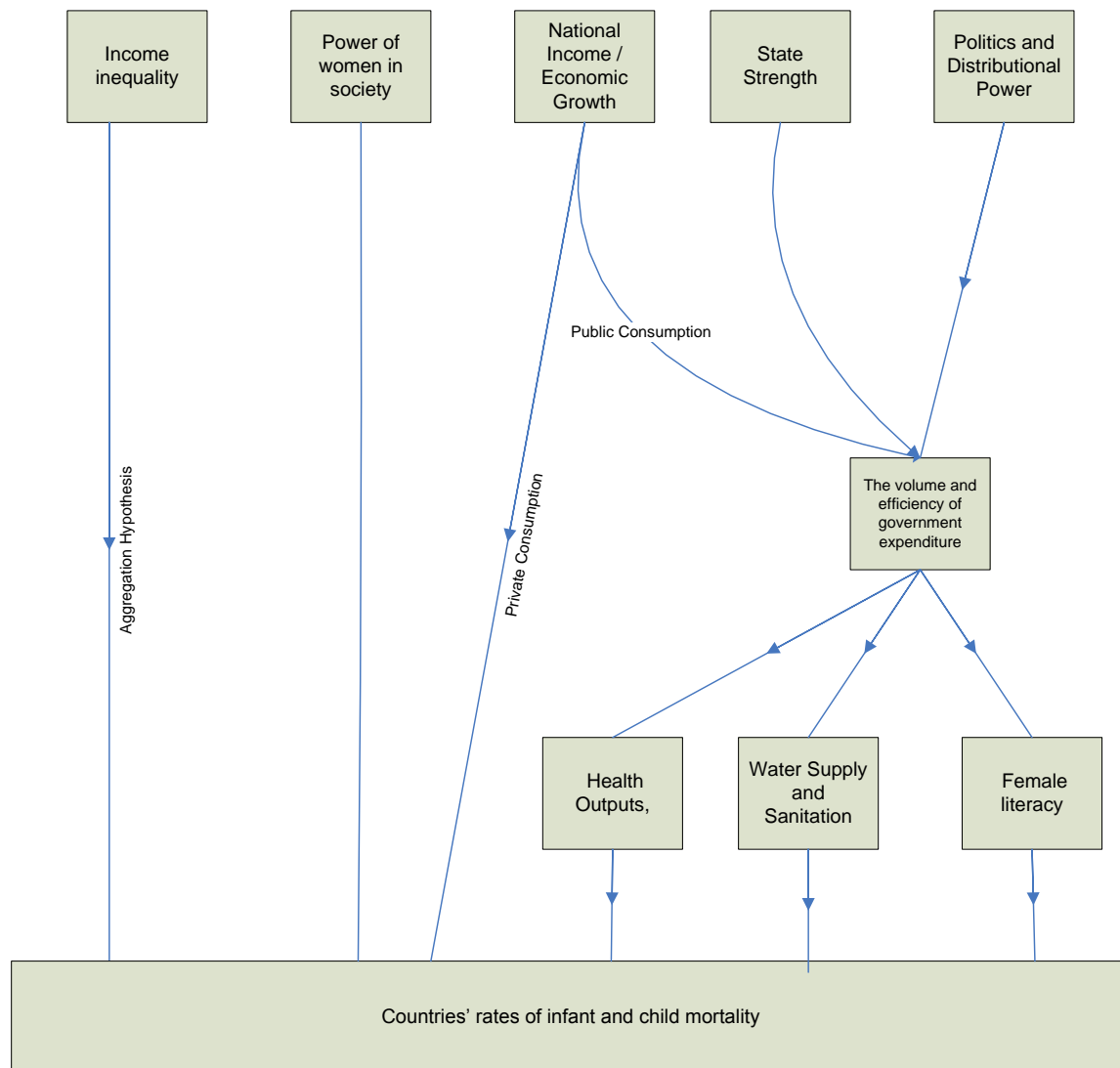
There is also indirect empirical evidence for the assertion that the distribution of power between groups influences mortality. Filmer and Pritchett (1999) found that ethnic linguistic fractionalization was a significant determinant of mortality. And Waldmann (1992) finds that income inequality, beyond that explained by its association with family income, partly determines child mortality.

We have argued that the power of different socioeconomic groups and state capacity may determine the efficacy of public spending on healthcare in reducing child mortality. The socioeconomic variables water supply and sanitation and female education are also to some extent outputs from public expenditure. So the distributional power of different socioeconomic groups and state capacity will also determine the efficacy of public spending in providing healthcare, water supply and sanitation and female education, and ultimately reducing mortality.

To summarize, we show the causal relationships between socioeconomic variables. For theoretical and empirical reasons the socioeconomic variables included in this framework

are: income, income inequality, female power, distributional power, public spending on social policy, state capacity, female literacy, health outputs and water and sanitation.

Diagram 2: The Socioeconomic Determinants Analytical Framework



National Income / Economic Growth

There is considerable evidence that increased income per capita reduces child mortality. The three pathways through which economic growth may affect child survival are private income, public provision and technocratic knowledge.

The private consumption mechanism states that increased national income per capita is associated with increased family income. Higher family income increases the consumption of goods and services that directly or indirectly improve child survival. Increased family income may also change behaviour. Richer families may, for example, place a higher value on their time. This can in turn lead to a reduction in time consuming activities, such as child rearing. The resulting lower fertility may improve birth spacing and reduce child mortality.

Increased national income per capita can also reduce child mortality through the public provision mechanism. The argument here is that economic growth increases the resources available for government expenditures on healthcare, education and infrastructure, which reduce child mortality.

Economic growth may also reduce infant and child mortality by stimulating technological innovation. This causal pathway is, however, not included in the diagram. The reason is that advances in medical technology are effectively outside of the control of the majority of developing countries; as they are principally determined by the private sector and government sponsored research in developed countries (Sumathipala et al 2004).

Income Inequality

Income inequality could be associated with lower rates of child survival through the aggregation hypothesis or the relative income hypothesis.

The aggregation hypothesis proposes that diminishing marginal returns between family income and child survival leads to a negative association between income inequality and child mortality at the national level (Rodgers 1979). Increasing a poor family's income from \$300 to \$450 decreases the chances of their children dying by more than increasing a rich family's income from \$4000 to \$4150 does. This implies that for a given income, the more unequal the mean deviation measure of income dispersion between families the higher the overall rate of child mortality. Because different measures of inequality are highly correlated, most measures of inequality can be substituted for the mean deviation measure of dispersion.

The aggregation hypothesis is a plausible explanation for the observed relationship between income inequality and child survival. Yet Waldmann (1992) has shown that even when the income of the poor is held constant, countries' child mortality rates increase with the share of national income held by the richest five percent of society. This result suggests the aggregation hypothesis is incorrect, because income inequality increases child mortality even when no group within society sees their actual income fall. This suggests that either income inequality actually causes poor child health (the relative income hypothesis) or that it is a proxy for another variable that does.

The two most commonly proposed pathways through which income inequality could cause poor child health are psychosocial effects and social capital. The psychosocial effects mechanism postulates that humans are envious creatures whose health suffers due to stress or related factors when they realize that they have a low income or position in society compared to others. Academics originally proposed this mechanism to explain the link between income inequality and life expectancy in developed countries (Marmot and Wilkinson 2004). More recent work by Wilkinson and Pickett (2009) has built on this hypothesis arguing that inequality erodes trust, increases anxiety and illness and encourages excessive consumption among the poor. This work shows that a wide range of health and social problems, including child well-being outcomes, are worse in rich unequal societies than in rich egalitarian ones.

The psychosocial mechanism can only explain the link between income inequality and child health if either children are aware of their social position, or else if their parents' knowledge of this affects their children's health. The applicability of this mechanism to developing countries where most children die from curable, well-understood non-stress related diseases

is also questionable (Jones et al 2003, Cutler et al 2006). This mechanism is, therefore, excluded from the analytical framework.

The social capital mechanism hypothesizes that income inequality extenuates actual or perceived differences in interest across individuals and social classes. This reduces trust, hampering cooperation and ultimately leading to government policies that only provide basic services to certain groups, increasing child mortality. It may also lead to an unjust social climate, limiting the poor's access to goods and services and further reinforcing income inequality.

The social capital mechanism argues that income inequality drives the distribution of services. Yet service provision is not principally determined by individuals relative wealth, but by their power to influence government. The relative wealth of different economic groups may partially determine their relative power, but other factors such as their ability to garner votes may also be important. It follows that multivariate studies may be finding that income inequality is significant, even when the absolute income of the poor is controlled for, because it is acting as a proxy for the power of different socioeconomic groups and the relative distribution of public services.

In conclusion, income inequality affects child survival through the aggregation hypothesis and not the relative income hypothesis. The relative income hypothesis is excluded because, in developing countries, neither the psychosocial effects nor the social capital mechanism are supported by a strong theoretical case or empirical evidence. Our econometric analysis empirically tests both these hypotheses.

Power/Status of Women in Society and the Family

Societies where women have greater prestige and influence may have higher rates of child survival (Caldwell 1986). This could be occurring because of women's power as a group to influence government policy. Yet this mechanism is not included in the analytical framework because it is hard to think of developing countries where the overriding source of political conflict is gender relations, more often politics is split along lines of class and ethnicity (Kohli 2004, Korpi 1983 and Migdal 1988).

A more likely explanation is that in societies where women have a high standing they have more control over their own family's resources and spending decisions (Caldwell 1986); leading to an increase in children's consumption and a subsequent reduction in their mortality. In societies and communities where women have higher standing they may also regard maternal and child healthcare as their right and demand and receive higher quality care from health professionals (Caldwell 1986). More powerful women may also be able to better negotiate the use of contraceptives with their partners; reducing fertility and child mortality.

There are strong theoretical arguments that mothers' power/status can affect mortality. There is also a complex relationship between mothers' status and their education. Societies in which women are of higher status may well be those where mothers can and do become educated. Female education could affect mortality independently of mothers' power by increasing their knowledge and use of specific health interventions and childcare practices (Caldwell 1986). Alternatively education may reduce child mortality by increasing women's power and status.

This complex interrelationship between status and education is represented in the analytical framework as follows. First, mother's education and women's power are included as separate socioeconomic variables. Second mother's education is seen as an output from government spending. Third, mothers' education increases their power. And fourth, women's power is associated with control over household resources and children's consumption. This specification effectively assumes that women's power does not drive educational attainment. Chapter 4 empirically tests the relative importance of women's power and education.

Politics and Distributional Power

Government health and education policies can potentially reduce child mortality. Politics may be regarded as the process by which socioeconomic groups influence government policy (Heywood and Gamble 2003). The power of different socioeconomic groups to influence government policy may vary. It follows that different distributions of power between socioeconomic groups may affect the formulation of social policy and its effectiveness in reducing mortality.

The argument that the power of different socioeconomic groups (distributional power) affects the formulation and effectiveness of social policy is strongly supported by many earlier historical/political economy studies such as Korpi (1983). Other authors have argued that the distribution of power between socioeconomic groups affects the development of modern state institutions which can implement effective public policies (Moore 1993 and Skocpol 1979). Likewise some authors have argued that there is a complex interaction between different types of state institution - such as whether or not a country is Federal or Democratic, the distributional power of socioeconomic groups and the efficacy of social policy²¹. Bonoli (2001) has argued with reference to developed countries that Federal Systems can hinder the development of welfare states and effective decision making. Some authors such as Navarro et al (2006) link the performance of health systems to political variables.

There is a strong theoretical argument and some non-econometric empirical evidence that the power of different socioeconomic groups may interact with and work through government institutions to affect the formulation and efficacy of social policy. Yet there is limited evidence from cross-country multivariate studies that such variables do affect mortality. There are five reasons for this.

First, the power of socioeconomic groups is a difficult concept to measure and no reliable cross-country data exist for this variable. This means it is difficult to include this variable in a regression analysis. The significance of ethnic-linguistic fractionalization variables and income inequality (even after controlling for a family's absolute income) in some multivariate studies can be read as providing indirect support for the argument that power of socioeconomic groups partly determines child mortality.

Second, the interaction between socioeconomic groups, government institutions and social policy may be inherently difficult to measure. It is difficult to capture such complex relationships using a regression analysis.

Third, there may be a divergence between a government's actual and stated policies. Peters et al 1999) argue, for instance, that although most African countries have stated that they are committed to prioritising the improvement of primary care, they were actually prioritising expensive tertiary health services. Similarly there is much evidence that although many developing countries have policy documents committing them to egalitarian health services, high-income groups often receive the most benefits from public health services (Victora et al 2003 and Gwatkin et al 2004).

Fourth, countries' institutions and political systems are difficult to classify without a detailed knowledge of that country. For instance some studies attempt to econometrically test whether democracy affects mortality (Navia and Zwiefel 2003). Many countries have some democratic elements but are not fully functioning democracies. Nigeria has for example elections, free speech and a free press but its elections are also marred by violence, voting based on ethnicity and payments to citizens to secure their votes (Obi 2001). So whether or not Nigeria should be classified as a democracy in a regression is complex.

Our analytical framework includes a box labelled "distributional power" to show the potential importance of these variables in determining mortality. Yet we recognize that it is difficult to test these variables using econometrics. Therefore while we do include variables to measure politics in our econometric analysis in chapter 4, we also examine a country which has low child mortality for its structural factors and briefly examine how politics and the distribution of power between socioeconomic groups underlay its effective social policies, in chapter 5.

State Capacity and Public Expenditure

A state's capacity refers to its ability to formulate and implement social policies. The analytical framework shows that a state's capacity partially determines social expenditures' effectiveness.

There is a strong a-priori case for assuming that the greater a state's capacity the more efficient its social policies and expenditure will be in reducing child mortality. Multivariate studies also offer some support for this, with three studies finding that government capacity reduces child mortality, whilst other studies fail to include state capacity, as opposed to finding it insignificant. There is also evidence from other cross-country and panel data econometric studies that state capacity is associated with other positive development outcomes such as improved water supply or sanitation or education. (Kaufmann et al 1999). This provides a second causal pathway through which state capacity may reduce child and infant mortality.

State capacity has a number of components. Some of the most important of these in terms of affecting the efficacy of public spending in achieving reduced mortality are the quality of budget and financial management, the quality of public administration, the education level of civil servants and the degree of corruption (Schell et al 2007). The WB's CPIA criteria measure these variables across countries. Therefore, we would expect countries with high state capacity, as measured by these variables, to make public expenditures that more effectively reduce child mortality.

Social Expenditure Outputs

Social expenditures produce outputs that reduce child mortality. Multivariate studies show that health outputs, female education/literacy and water and sanitation all significantly reduce child mortality, and therefore these outputs are included in the analytical framework. Our definition of health outputs includes outputs from family planning programmes such as the availability of contraceptives. Effective family planning programmes can reduce fertility and increase birth spacing (Cleland et al 2006).

Hypotheses

The socioeconomic analytical framework leads to the following testable hypotheses:

- GDP per capita, income inequality and women's power should explain much of the variation in child survival across countries and time;
- The impact of public spending on health in reducing child mortality across countries may be low, but there should be a subset of countries with high state capacity where public expenditure on health is relatively effective in reducing mortality;
- Public expenditure may be ineffective and inefficient in reducing infant and child mortality across countries, but there will be a subset of countries with amenable politics and high state capacity where public expenditure will be effective;
- Health outputs will be higher per unit of expenditure in countries with high state capacity and effective politics.

Interlinking Socioeconomic and Proximate Determinants

Diagram 3 shows how different socioeconomic factors affect child mortality through proximate variables. It demonstrates that increased family income can reduce child mortality through three mechanisms. First, richer families can afford to feed their children more and better quality food reducing nutrient deficiency. Second, family income may also be associated with parents valuing their time more and deciding to have fewer children; leading to improved maternal factors such as a better birth spacing. And third, higher income may improve personal illness control by allowing families to purchase better medical care and by removing barriers such as transport costs and user fees to medical services.

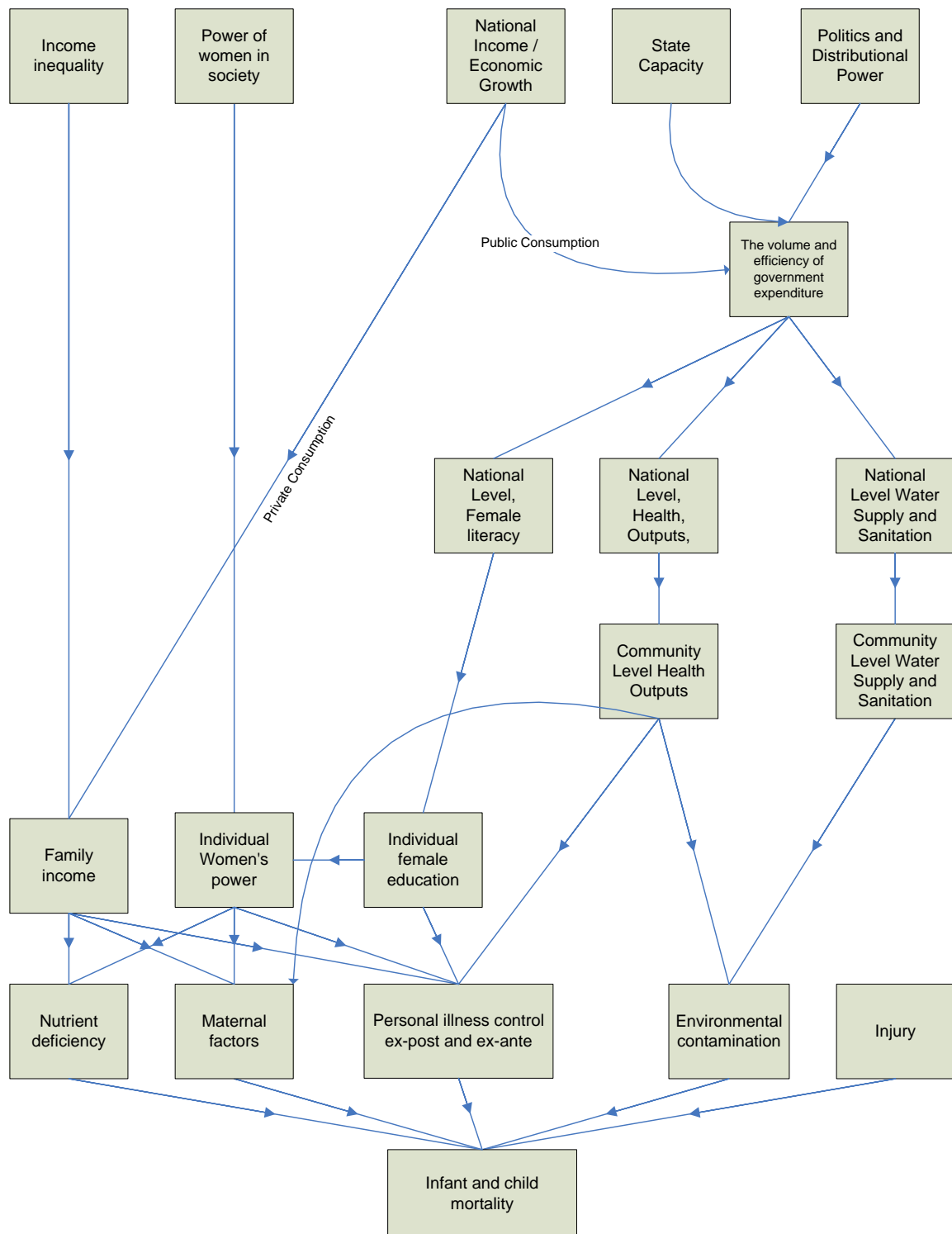
The power/status of a mother within the family can also reduce child mortality. An increase in the mother's power relative to other family members often leads to an increase in the resources spent on children, raising their consumption on food and medical goods and hence improving nutrition and illness control. More powerful women may also have greater control over birth control decisions causing an improvement in maternal factors through reduced fertility.

Female education indirectly reduces child mortality by increasing the mother's power within the family. It directly improves child survival by increasing knowledge of health interventions and illness control.

Health outputs at the community level are associated with improved personal illness control, for example, by improving the chances that ill children will be taken to a doctor, correctly diagnosed and receive appropriate treatment. They are also associated with reduced environmental contamination, by, for example, vaccinations reducing the overall prevalence, and thus any individual's chances of contracting a particular disease. Health outputs are also associated with maternal factors because outputs from family planning programmes, such as access to contraception, can affect birth spacing and the age of the mother at birth.

Water supply and sanitation is seen as reducing environmental contamination from diseases such as cholera.

Diagram 3: Final Analytical Framework



Conclusion

This chapter started by arguing that simply postulating a statistical relationship between socioeconomic variables and child survival is insufficient. In order to investigate policy, the causal pathways through which socioeconomic variables affect child survival must be established and discussed. This chapter outlined these causal pathways through a detailed and comprehensive analytical framework of the determinants of child survival. This framework was built on two assumptions. First, socioeconomic variables cannot directly affect child survival but must instead work through proximate variables. And second, some socioeconomic variables work through other socioeconomic variables in affecting proximate determinants.

Building on earlier work, the analytical framework then established a comprehensive list of proximate determinants, and showed how these interacted with each other. The most important testable conclusions from the proximate determinant framework were that a) all socioeconomic determinants and proximate determinants are potentially significant in explaining infant and child mortality whatever the registered cause of death and b) that the number of deaths recorded as occurring due to a particular disease is not necessarily the same as the actual number of deaths caused by that disease.

This chapter, using empirical evidence from earlier multivariate studies, created an analytical framework of the socioeconomic determinants of child mortality. It showed how income, income inequality and women's power influence mortality through a range of proximate variables. We argued that distributional power and state capacity determine the efficacy of public spending in reducing child mortality. We use the analytical framework outlined here to guide the empirical analysis contained in the next chapter. More specifically, a panel data econometric model is used to test the hypotheses outlined earlier in this chapter.

Chapter 4: Statistical and Econometric Analysis

Introduction

This chapter analyzes the socioeconomic causes of child mortality. This analysis is guided by the analytical framework and literature review discussed in previous chapters.

This chapter is divided into three main sections. The first discusses child survival data from WDI and DHS datasets. The second section constructs a cross-country model of the socioeconomic causes of survival mortality. This tests whether, as hypothesized by our analytical framework, income, income inequality and women's power are significant determinants of child deaths and under-nutrition across countries. A panel data model of child mortality is constructed in the third section. We use this to test the following hypotheses specified by our analytical framework:

- Income, income inequality and women's power are determinants of child survival;
- Income, income inequality and women's power are determinants of child under-nutrition;
- It is women's power, not their education, which mainly determines child survival;
- Income inequality determines mortality through the aggregation, and not the relative income, causal pathway;
- Politics and state capacity determine the effectiveness of public spending in reducing child survival.

Data

The majority of econometric studies use child mortality data from international datasets such as WDI, UNICEF dataset or the MDG database. These are comprehensive including data for nearly all countries in all recent years mainly because the donor and academic communities demand such data²². Yet most developing countries lack the systems necessary to collate yearly child mortality data. Developing countries' statistical offices mainly collate child mortality data from two primary sources — vital registration systems and censuses. In many developing countries the vital registration system does not accurately record all births and under five deaths and is therefore an inaccurate source of information with significant measurement error (UN 1992). Many, but by no means all, developing countries are able to undertake censuses, but these are only carried out every five or 10 years (Wang 2002).

²²Murray et al (2007) discuss the problems with the current international databases in some detail. This study creates a potentially more accurate dataset by merging available databases and adding to them where possible. This dataset may well be the most accurate dataset available which covers all, or nearly all, countries. But Murray et al (2007) cannot escape the problem that monitoring MDG4 requires accurate yearly child mortality data which most developing countries cannot collect. There is a trade off between accuracy and comprehensiveness. Our analysis examines all countries using WDI data and then restricts itself to those countries for which accurate and comprehensive data exist by using our own databases based on adjusted DHS data.

The international datasets, therefore, use data collected via vital registration systems, national censuses, multiple indicator clusters and DHS. In addition, for some countries and years actual data do not exist, so the reported under-five mortality rate is predicted based on past trends (Murray et al 2007). The original source of data for the WDI and MDG databases is also very difficult to trace; with the MDG database classifying the source of data for many countries as “estimated”, which means:

“The figure is estimated by the international agency, when corresponding country data on a specific year or set of years are not available, or when multiple sources exist, or there are issues of data quality. Estimates are based on national data, such as surveys or administrative records, or other sources but on the same variable being estimated”

The above statement does not clearly elucidate what the actual source of these data was! Rather it clearly shows that there is a much higher demand for data on child mortality than there is the ability by developing countries to accurately produce it.

The inclusion in international databases of data from different sources and interpolations causes two problems. First, time series analysis based on these data is likely to predict trends which are determined by the rules and assumptions governing the original interpolations and not the true rate of decline or increase in child mortality (Bhargava et al 2001). Second, different sources give different estimates of under-five mortality. This suggests that data from different sources may not be strictly comparable across countries or periods²³.

DHS provide an alternative source of mortality data. Macro-International has carried out surveys in 70 countries, and in many countries they have been undertaken for more than one period. DHS have standardized survey instruments for all countries and the methods they use for sampling and data collection are reasonably similar across different periods. These surveys can be used to accurately compare child mortality rates across countries (Sahn and Stifel 2003).

The main problem with using DHS to examine trends in child mortality is that they are undertaken at irregularly spaced intervals and that some countries only have one survey. Macro-International does calculate past rates of infant, under-three and under-five mortality from DHS, but only data for five year averages is publicly available²⁴.

A further problem with using DHS data is that while the survey includes children and infants with mothers aged 15 to 49 at the time of the survey, the average age of children’s mothers will decrease as mortality rates are calculated for earlier periods (Sahn and Stifel 2003). For example, data from ten years prior to a survey will not include mortality data for children born to mothers aged 40 to 49 at the time of the child’s birth. It follows that if a mother’s age is a significant determinant of infant mortality, and there is evidence in DHS data that it is, then the further back in time infant and under-five mortality rates are calculated the larger will be the downward bias in these rates (Sahn et al 1999). Thus these rates will not be comparable within countries across time.

We avoid this bias by recalculating infant and under-three mortality rates from individual mothers’ responses to DHS in 61 developing countries. Under-five mortality rates are not

²³ Author’s own calculations, results not reported.

²⁴ See: Measure DHS at: <http://www.measuredhs.com>

calculated because this would lead to just five years of data per survey, but as we would expect a very strong correlation between under-five and under-three mortality this is not a major problem. Our dataset uses a truncated sample that only includes children from mothers aged 15 to 39 at the date of the child's birth and only calculates mortality data for the 10 years preceding the survey. Infants born one year prior to the survey are excluded from this analysis as these children may have died in their first year but after their mother was interviewed. For the same reason under-three mortality data was calculated for seven years. In summary, this technique gives us seven years of under-three mortality data and nine years of infant mortality data for each survey conducted in 61 countries. Data are comparable both across countries and across time and represent the most comprehensive and accurate data available on child mortality²⁵. Furthermore no previous econometric analysis has used this dataset. This dataset is referred to as DHSadj (adjusted) throughout the rest of this thesis. The term DHS refers to the mortality data calculated by Macro-International. Our statistical analysis proceeds by using DHSadj (for accuracy), WDI (for comprehensiveness; so that we can include more countries) and DHS to check our results²⁶.

A Cross-Country Model of the Structural Determinants of Child Survival

This section constructs an econometric model to explain cross-country variation in child survival. Prior to specifying this model, we discuss the measurement of the independent and dependent variables, the appropriate lag structure and the correct functional form.

Measurement of Independent Variables

Our dependent variable (child survival) is measured through neonatal mortality (DHS), post neonatal mortality (DHS), infant mortality (DHS, DHSadj and WDI), under-three mortality (DHS and DHSadj) and under-five mortality (WDI and DHS). We also include under-nutrition (WDI) as an independent variable as this is a good measure of overall health status and should be closely related to child survival. In order to smooth out yearly variations in the child survival and under-nutrition data, and because DHS data are publically available in five year averages, we therefore follow existing research such as Pritchett and Summers (1996) in using five year averages of the independent and dependent variables.

This chapter uses GDP per capita at purchasing power parity in current US Dollars to measure income. The purchasing power parity measure is used because the cost of many of the goods consumed by families, and health inputs purchased by governments, substantially vary between countries.

This paper uses the ratio of girls to boys in secondary education as a proxy for women's power. The advantage of this measure is that it is available for a wide range of countries. It may, however, be correlated with female education — societies where a high number of girls are educated relative to boys may also have high absolute levels of female education. This is

²⁵ The total number of surveys that these data were calculated for does not equal the total number of DHS carried out because access to some surveys' datasets is restricted by Macro-International.

²⁶ Appendix 3 contains a statistical analysis comparing WDI and DHS datasets

problematic because there is a reasonable theoretical argument and some evidence for female education reducing child mortality; meaning that any significant coefficient in our dependent variable could be picking up the importance of female education.

Because of this problem we examined other measures of women's power. Other measures that were considered were: the proportion of seats in the national parliament held by women, the female labour force participation rate, proportion of economically active female children and the adolescent fertility rate. We did not in the end use these measures of women's power for three reasons.

First, all these measures were themselves highly correlated with measures of female education. The use of these measures as a proxy for women's power would not solve the underlying problem that this independent variable could be picking up the impact of female education on mortality.

Second, there are also theoretical problems with these measures of women's power. The proportion of seats in the national parliament held by women may not be a true reflection of their power in society. The reason for this is that in many developing countries the legislature holds limited real power and women's participation in parliament may reflect the image developing country governments want to display internationally and, thus, not accurately demonstrate the true status of women in society. The female labour force participation rate, meanwhile, is related to female education because more educated women are more likely to be employed in the formal sector. Likewise the proportion of economically active female children is related to school attendance as children cannot be both economically active and attend full-time education. The introduction of the adolescent fertility rate into our model would be problematic because a mother's age at their child's birth is a direct proximate determinant of mortality.

There is also a complex interrelationship between women's power and their education. Access to education in some societies may have precipitated an improvement in women's status and control over resources. In contrast, in other countries women's relatively high status may have precipitated their access to education. This means that across countries measures of women's education and power are always likely to be highly correlated and that searching for a measure of women's power that is theoretically and empirically unrelated to female education is unlikely to be fruitful.

Third, the use of the ratio of girls to boys in secondary education, as opposed to other measures of women's power, leads to a higher sample size in our panel data econometric models. So, in summary, we use the ratio of girls to boys in secondary education as a measure of women's power because it is available for a large number of developing countries and because other measures of women's power are also theoretically and empirically related to female education. We are, however, aware that our measure of women's power may be affecting child survival because it is correlated with female education. Later in this chapter we further investigate the relationship between women's power, female education and under-five mortality by testing the significance and explanatory power of models that include different measures of female power and education²⁷.

²⁷ We also checked our results by using the proportion of seats in the national parliament held by women as a proxy for women's power and rerunning the models contained in tables 8 and 9 of this chapter. This did

We measure income inequality through the Gini index. This is a statistical measure of inequality ranging from zero (perfect equality, everybody has the same income) to 100 (perfect inequality, one person has all of a society's income).

Lag Structure

Some multivariate studies include a five year lag between the independent variables and child mortality (Shen and Williamson 2001) but some do not include any lag (Jamison et al 2006 and McGuire 2006). The logic behind using a five year lag of income is that income may only slowly translate into increased government spending and better health services. However, as increased GDP per capita is also associated with families' income which should almost immediately reduce mortality; there is also a strong case for including a shorter or no lag. As earlier multivariate studies concluded that income mainly affects child mortality through family income (Filmer and Pritchett 1999), we assume that income determines child survival without a lag.

Women's power could affect child survival either immediately or with a five year lag. It could almost immediately affect mortality if it primarily reduces child survival by increasing women's control of resources. Alternatively, if it affects child survival either by reducing fertility or by increasing access to education, or other public services then we would expect it to affect mortality with a lag. Our analytical framework hypothesis is that women's power affects child survival mainly by increasing mothers' control over household resources, so our model does not lag this variable.

Income inequality is also modelled without a lag. The reason for this is that the theoretical framework shows that income inequality affects child mortality through its relationship with family income; meaning that it should almost immediately affect child mortality. Alternatively if income inequality represented the power of different socioeconomic groups to influence government policy then we would expect it to affect mortality with a lag.

Functional Form

This chapter initially estimates all models using the double log functional form. There are three reasons for using this functional form.

First, as discussed earlier, because child mortality is bounded at zero there are strong theoretical reasons for considering the relationship between it and income will be non-linear and this is recognized in the literature with many models using the double log functional form.

Second, the bivariate relationship between income and under-five mortality (see appendix 5) shows that as income increases, under-five mortality declines, but at a decreasing rate²⁸. This

not lead to a substantial change in the results of our models or the conclusions drawn. More specifically, income, income inequality and women's power continued to be highly significant for different measures of child survival with a high R-Squared.

²⁸ The graphs in the appendix are for under-five mortality (WDI and DHS). Graphs for the other measures of child survival show a similar relationship. This can be most clearly seen in the graph using WDI data. The relationship is not as clear for DHS data because these surveys are mainly conducted in poorer developing countries.

suggests that either a linear log or double log functional form should be used. A boxcox²⁹ regression also provided strong evidence for the double log formation. The bivariate relationships between income inequality, women's power and under-five mortality shown in appendix 6 are more difficult to interpret³⁰.

Third, the double log functional form gives results in elasticities, which are easy to interpret³¹. Other functional forms are used to check the robustness of our models.

The model we are estimating is thus:

$$\ln Y_i = \beta_0 + \ln \beta_1 X_{1i} + \ln \beta_2 X_{2i} + \ln \beta_3 X_{3i} + U_i$$

When Y = a measure of child survival, and the three independent variables are income, income inequality and women's power.

The results in table 3 offer strong support for our hypothesis. Across countries, income, income inequality and women's power significantly determine child survival as measured by post-neonatal mortality (DHS), infant mortality (WDI, DHS), under-three/child mortality (DHS, and DHSadj) and under-five mortality (DHS and WDI) and under-nutrition (WDI). These results are summarized in table 2.

Table 2: Summary of Results from Cross-Country Econometric Model

Variable	Expected Sign on Coefficient	Significant at 10% level	Significant at 5% level	Significant at 1% level	Not Significant in which Models at 10% level
Income	Negative in 10/10 models	9/10 models	9/10 models	8/10 models	DHS Neonatal
Income Inequality	Positive in 10/10 models	8/10 models	6/10 models	5/10 models	DHSadj infant and DHS neonatal
Women's Power	Negative in 10/10 models	10/10 models	8/10 models	7/10 models	None

²⁹ See: Box and Cox (1964).

³⁰ Appendix 6 shows the bivariate relationship between women's power and under-five mortality, and income inequality and under-five mortality. Higher women's power is associated with lower mortality. And higher income inequality may be associated with higher mortality. Bivariate graphs were also drawn but are not reported for other measures of child health and with different lag structures. All graphs gave similar results.

³¹ All results were checked for robustness using other functional forms.

The high significance levels on most of the coefficients, the consistency of the results across different datasets and the high adjusted R-squared ranging between 0.491 (under-nutrition WDI) to 0.818 (WDI infant mortality) demonstrate the robustness of these results. Similar results were also produced using a 10 year average of all the variables, using five year lags and using a linear specification³². There is, therefore, no reason to believe that these results occurred by chance.

There are, however, two outlying results. First, income inequality is not significant for infant mortality using DHSadj data. This result is probably occurring because of the small sample size in this regression which includes just 28 observations. The significance of income inequality in the regressions where infant mortality is measured using DHS and WDI data provides strong evidence that this variable does significantly determine infant mortality across countries. Second, neither income nor income inequality are significant in determining neonatal mortality. This may be because family income affects infant and child mortality through its relationship to the quality of food and medical interventions families can afford. These proximate variables may not affect neonatal mortality, which is more strongly related to genetics at birth and maternal factors, such as birth spacing, birth order and mother's age at their birth, than it is to neonates' ex-post birth consumption. The high significance of the women's power variable and its relatively large coefficient with reference to neonatal mortality may indicate the impact this variable has on fertility, birth spacing and mother's age at their child's birth. Simply put, when children die at a very young age there may be less time for differences in family income to affect mortality.

In conclusion, our cross-country model provides evidence that income, income inequality and women's power are significant determinants of under-nutrition, post-neonatal mortality, infant mortality, under-three mortality and under-five mortality.

³² Results not reported.

Table 3: Cross-country Model of the Determinants of Mortality (nutrition stands for growth faltering/under-nutrition)

Cross-country OLS regression with natural logs of dependent and independent variables and no lags (infant mortality (DHS) and under-five mortality (DHS) have robust standard errors to account for hetroskedasticity)

Column	1	2	3	4	5	6	7	8	9	10
	WDI Under-Nutrition	WDI Infant	WDI Under-five	DHS Neonatal	DHS Post Neonatal	DHS Infant	DHS Under-three	DHS Under-five	DHS Adj Child	DHS Adj Infant
GDP Per Capita, PPP (current US\$)	-0.686**	-0.721**	-0.792**	-0.161	-0.477**	-0.323**	-0.680**	-0.428**	-0.463**	-0.279*
Gini Index	0.822+	1.036**	1.077**	0.373	1.634**	0.978*	1.983**	1.320**	0.988+	0.285
Ratio of girls to boys in primary and secondary education	-1.407+	-0.689+	-0.908*	-1.610**	-1.585**	-1.574**	-3.469**	-2.147**	-2.179**	-1.931**
Constant	11.15**	8.716**	10.42**	10.48**	7.884**	9.912**	16.51**	12.37**	13.95**	13.90**
N	53	69	69	31	32	32	32	32	26	28
Adjusted R-Squared	0.491	0.818	0.835	0.530	0.637	0.646	0.753	0.744	0.585	0.547

Key if + p < 0.1 *p < 0.05 ** p < 0.01

A Panel Data Model of the Structural Determinants of Child Mortality

Introduction and Tests of Convergence

The previous section examined the determinants of child survival across countries. The use of a cross-country model avoided some of the more complex statistical issues such as stationarity and auto-correlation that are common in time series and panel data models. But restricting the data to a single period led to a small sample size (26 to 31) for DHS and DHSadj data. The use of a cross-country model also meant that we could not examine trends in child survival across time.

We ameliorate these problems by using panel data. This includes data for different countries and within those countries for different periods. This panel dataset allows us to question whether there has been a convergence in child death rates. Have countries with initially high child death rates improved their children's health faster than those countries with initially low child death rates? Is the distribution of child death more egalitarian now than in the past?

We answer this question by regressing the first difference of the child survival and under-nutrition variable on its one period lag and a series of five year time dummies. A negative coefficient on the independent variable would illustrate absolute convergence unconditional on anything except for time. The equation for this analysis is:

$$dY_{it} = B_{0t} + \beta Y_{it-1} + U_i$$

When Y = a measure of child survival or under-nutrition

The results, displayed in table 4³³, demonstrate unconditional convergence. The coefficient on the lag of the dependent variable is negative and significant (at the 5% level or less) for every measure of child survival and under-nutrition. This shows that child deaths across countries are becoming more egalitarian. But this convergence is only occurring slowly over time. Even for neonatal mortality, which shows the highest rate of convergence, a country which has an extra neonate death at the beginning of the five year period will on average only see an extra 0.2 unit reduction in deaths over five years. At this rate it will take many years for countries with poor child survival to catch up to those with better child survival.

Younger (2001) argues that if the beginning of period infant or child mortality rate is measured with considerable error then the results may suffer from a bias towards zero. Younger accounts for this bias by instrumenting the dependent variable on its lag, but finds that this does not significantly affect the results. We do not use instrumental variables here both because Younger does not find that it significantly affects his results and because our DHSadj data should measure the beginning period infant and under-three mortality rate accurately.

³³ These results were calculated using five year averages of child health. Results, not reported, using one year data showed a similar story.

Table 4: Convergence over Time across Countries for Different Measures of Child Survival and Under-nutrition

	WDI Nutrition	WDI Infant	WDI Under-five	DHS Neonatal	DHS Post Neonatal	DHS Infant	DHS Child	DHS Under-five	DHS Adj Child	DHS Adj Infant
Lag of Dependent Variable	-0.127***	-0.0448***	-0.0461***	-0.205***	-0.115***	-0.119***	-0.111***	-0.0881***	-0.0603*	-0.0958***
1965/1969 Time dummy	0	0	0	0	0	0	0	0	0	0
1970/1974 Time dummy	0	-0.788	-1.074	0	6.966*	0	0	0	0	0
1975/1979 Time dummy	0	-1.988*	-2.682	0.421	3.717	-2.402	1.914	-0.332	0	0
1980/1984 Time dummy	0	-2.640**	-4.186**	1.741	2.316	-2.652	-1.399	-3.570	0	6.903
1985/1989 Time dummy	8.001**	-0.191	0.956	-0.375	1.565	-4.686	-1.100	-4.907	-1.140	3.225
1989/1994 Time dummy	8.930***	1.972*	4.596**	-0.64	2.953	-3.925	0.483	-2.350	4.903	8.045*
1995/1999 Time dummy	9.401***	3.871***	7.689***	0.349	4.742	-0.272	2.996	3.595	8.258	9.783**
2000/2004 Time dummy	8.142***	3.541***	7.278***	-1.188	2.229	-4.310	3.501	0.231	-4.184	0
2004-2006 Time dummy	8.780**	4.228***	8.152***	-8.073**	0	-13.83***	-3.240	-14.31*	0	0
Constant	-7.536**	-5.641***	-10.33***	5.134**	-2.275	5.392	-1.895	-0.668	-6.866	-6.533
N	185	1186	1176	365	370	373	370	371	161	181
Key	* p<0.05	** p<0.01	*** p<0.001							

Table 4 also shows that unconditional convergence is higher for DHS than WDI data. For under-five mortality the coefficients are -0.0881 and -0.0461 respectively for DHS and WDI data. This difference may be occurring because the WDI and DHS datasets contain different samples of countries. Table 5 shows that the average value for income is higher, and under-five mortality is lower, in the WDI than DHS dataset. The faster unconditional convergence in DHS data might be showing that there was higher convergence between poor developing countries than between all countries (excluding upper-income countries).

Table 5: Differences in WDI and DHS Datasets for Average Income and Under-five Mortality

	WDI	DHS
Average Income (GDP Per Capita PPP) when under-five mortality data point exists	4,260.53	1,948.147
Under-five mortality	112.0562	138.5813

This thesis formally tests this hypothesis by rerunning the convergence regression for low, lower-middle, middle and upper-middle-income country groups (WB definition of income used). The results for this regression (see table 6) illustrate that in-group convergence was highest for countries in the lower-middle or middle-income groups. In-group convergence was lower in the upper-middle and low-income groups.

Table 6: Convergence in Under-five Mortality between Different Income Groups

	Under-five mortality WDI				
	All	Low Income	Middle Income	Middle Income	Upper Mid
Lag of independent variable	-0.0461***	-0.0345***	-0.139***	-0.118***	-0.0539***
1965/1969 Time dummy	0	-6.899**	0.864	0	-4.390
1970/1974Time dummy	-1.074	-7.676**	0	0	-6.433
1975/1979Time dummy	-2.682	-9.850***	-3.528	0	-3.388
1980/1984Time dummy	-4.186**	-9.934***	-5.993	22.59***	-7.118
1985/1989Time dummy	0.956	-5.588*	-2.575	30.82***	-2.013
1989/1994Time dummy	4.596**	-0.777	-0.566	33.77***	0
1995/1999Time dummy	7.689***	1.957	1.501	36.00***	7.256
2000/2004Time dummy	7.278***	0.302	2.735	35.33***	5.362
2004-2006Time dummy	8.152***	0	3.574	38.54***	6.067
_cons	-10.33***	-5.072*	-1.939	-37.10***	-5.698
N	1176	516	311	103	246
adj. R-sq	0.297	0.177	0.473	0.686	0.345

Key * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$

Tests of Stationarity

The rest of this thesis examines the determinants of child survival and under-nutrition across countries and time. Before we can begin this analysis, we first need to examine the trends in our variables over time. The reason for this is that if child survival is consistently decreasing over time (non-stationary) but one of the independent variables is moving around a consistent mean (it is stationary) then there cannot be a genuine relationship between them. Likewise, if two variables consistently increase over time then any long term relationship between them may be occurring simply because they both followed similar trends and not because one variable caused the other to change (spurious regression). The only exception to this would be if short term changes in one variable led to short term changes in the other variable (cointegration). In short, if child survival is stationary then its determinants must also be stationary. However, if child survival is non-stationary then its determinants must also be and the dependent and independent variables should also be cointegrated.

There are strong theoretical reasons for concluding that all our measures of child survival are stationary. All our measures of child survival must be stationary in the long term because they record the number of deaths occurring per 1000 births. All our measures of child survival are, thus, clearly bounded at zero and over the very long term we would expect mortality to approach this value and for its very long term average to be close to zero. Child malnutrition should theoretically also be stationary because it is bounded between zero and 100, and it cannot therefore have a mean which consistently decreases or increases over the long term.

Theoretically the measures of women's power and income inequality we have used should also be stationary. This thesis measured women's power as the ratio of girls to boys in primary and secondary education, which is clearly bounded between the value zero and 100. The Gini index which was used to measure inequality is also by definition bounded between two values.

The only variable that we would expect to be non-stationary is income. There is no reason to believe that our measure of income - GDP per capita in Purchasing Power Parity - is stationary over time. It could be that in the long term people's average income continues to increase. There are, however, good reasons to believe that transformations of the income variable will be stationary. Yearly percentage change in income, for example, would probably be stationary as there is no reason to believe that rates of economic growth consistently increase over the long term. Rather, many developed and upper-middle-income countries show consistent growth rates over long periods of time; suggesting that developing countries with very high rates of growth will not be able to continue increasing these, but will instead see growth rates stabilize or decline as they become richer. The natural logarithm of income may also be stationary.

Each variable was tested for stationarity using the Xtfisher test for unbalanced panels³⁴. The results from this test show (see appendix 9)³⁵ that all our independent variables apart from

³⁴ This test combines the p-values for independent unit root tests. It assumes that all series are non-stationary under the null hypothesis against the alternative that at least one series in the panel is stationary.

³⁵ The results from table 10 are for five year averages. Similar results were found but are not reported for five year average and one year data.

income are stationary. Both the natural log of income and the percentage change in income were, however, stationary. Some of the natural logs of the child survival variables are also non-stationary. Yet there is no theoretical reason for believing that these variables are non-stationary. Furthermore, it is implausible that for two closely related measures of child survival such as neonatal and post-neonatal mortality one could be stationary and the other non-stationary. For these reasons, we proceed as if all variables (apart from income) are stationary.

Panel Data Model of Income, Income Inequality and Women's Power

The inclusion of countries and years means there are three types of variation in child survival that our model could seek to explain. First, there is variation in a particular period across countries — why did China have lower infant mortality than Kenya in 1999? Second, there is the variation within countries over time — why did China's rate of infant mortality decline between 1990 and 2005? And, third, there is the average variation in mortality across time which is occurring for reasons other than that explained by changes in the determinants of child mortality in individual countries — why did the world's mortality decline at a faster rate than is explained by the determinants of the decline in individual countries' child mortality rates?

The exact specification of our panel data model depends on the type of variation we are trying to explain. The analytical framework outlined earlier provides a guide to the type of variation in child survival that we are seeking to explain. It does not differentiate between countries or periods. The analytical framework does not say that increased income would reduce child mortality in the 1980s but not in the 1990s. And it does not say that increased income will reduce child mortality in Kenya but not India. That is, it hypothesizes that the causal factors it includes should explain changes in child mortality over time, and between countries at a point in time. This suggests that we use a pooled model. We, however, include time effects to account for reductions in child mortality due to exogenous factors, such as changes in medical knowledge driven by research in OECD countries which are not included in our dataset.

This chapter rejects fixed and random effects models. Such models include a dummy variable to explain why some countries have higher child mortality than others. This normally improves the models explanatory power in a purely statistical sense because the dummy variable absorbs some of the unexplained variation in child survival but it also reduces their true explanatory power. For they tell us that one country has higher mortality than another, but not why! If there were other variables that we believed explained differences in child mortality across countries, then we would have included these in our analytical framework and empirically tested them.

This chapter also rejects unrestricted models because there is no reason to consider that the relationship between child health and its determinants varies across countries simply because of a countries' name or geographic location. The relationship between child mortality and a causal factor may vary due to a third factor. Our analytical framework for instance hypothesized that social policies' impact on child mortality varies according to state capacity. We analyze this relationship using interaction terms later in this chapter.

Our first panel data model includes income, income inequality and women's power as determinants of child health. The equation for this model is:

$$\ln Y_{it} = \beta_{0it} + \ln \beta_1 X_{1it} + \ln \beta_{2it} X_{2it} + \ln \beta_{3it} X_{3it} + U_{it}$$

This model³⁶, see tables 7 and 8, demonstrates that income, income inequality and women's power determine child survival and under-nutrition. These three variables are significant determinants of all our measures of child survival apart from neonatal mortality and infant mortality (from the DHSadj dataset), for which income inequality is insignificant. The coefficients on all three variables are of the expected sign. That is income and women's power reduce, and income inequality increase, mortality. These models also have significant explanatory power; explaining between 68% and 93% of the variation in the dependent variables.

Table 7: Summary of Results from Panel Data Model

Variable	Expected Sign on Coefficient	Significant at 5% level	Significant at 1% level	Significant at 0.1% level	Not Significant in which Models at 5 % level
Income	Negative in 10/10 models	10/10 models	10/10 models	10/10 models	N/A
Income Inequality	Positive in 10/10 models	8/10 models	7/10 models	5/10 models	DHS Neonatal Mortality and DHSadj Infant Mortality
Women's Power	Negative in 10/10 models	10/10 models	10/10 models	9/10 models	N/A

The results show that the elasticity of income ranges from -0.254 for neonatal mortality to -0.871 for under-five mortality DHS. This is broadly in line with the results found by earlier multivariate studies. Kakwani (1993) uses functional forms that allow for varying income elasticity in cross-national data and finds a range of between -0.5 to -0.6. Pritchett and Summers (1996) find a long run elasticity of between -0.43 to -0.76 (depending on the instruments used). Jamison et al (2006) find an elasticity of 0.65. The coefficients on income in our results are also significant at the 0.1% level for every measure of child survival and under-nutrition across all three datasets (DHS, DHSadj and WDI). The results therefore provide strong evidence that increased income improves child survival.

Our results also show that income elasticity is higher for under-five mortality (-0.782 WDI and -0.540 DHS) than it is for neonatal mortality (-0.254). This intuitively makes sense as family income and the goods it can purchase have five years to affect under-five mortality

³⁶ Results were corrected for autocorrelation using Panel Corrected Standard Errors. See appendix 11 for autocorrelation tests.

but relatively little time to affect neonatal mortality. In addition, neonatal mortality may be mainly determined by innate genetic factors; while income and the amount children consume may have a direct and larger impact on under-five mortality.

Regarding income inequality our result shows an elasticity of 0.845 for under-five mortality WDI. This is broadly in line with that reported in previous multivariate studies; with Flegg (1982) reporting an elasticity with respect to infant mortality of 0.77 and Pritchett and Summers (1996) reporting an elasticity of 0.51 with respect to under-five mortality.

The consistency of our results was confirmed by rerunning the panel data model using a linear model whereby only income is logged and by lagging the independent variables by five years (tables 9 to 11). This means that we have 40 regressions³⁷, because we have two functional forms (linear-log and double log), two lag structures (no lag and five year lag) and 10 measures of child survival (including under-nutrition). Income and women's power significantly improve child survival in 100% and 95% of these regressions respectively. And income inequality significantly worsens child health in 80% of them. Taken together these models provide overwhelming evidence that our original hypothesis was correct and that our results did not occur by chance or through the deliberate selection of a particular lag structure, functional form or dependent variable. So, in conclusion, income, income inequality and women's power determine child survival across countries and over time.

The results do, however, also show that most of our three dependent variables are strongly correlated with their lags. It is, thus, empirically problematic to determine which particular lag structure, zero or five years or something in between, is correct. This thesis favours the model with a zero lag structure for the theoretical reasons discussed earlier.

³⁷ We also rerun our original model using GLS to account for autocorrelation (see appendix 8).

Table 8: OLS Panel Data Regression with PCSE, Natural Logs of all Variables and No Lags

Panel OLS regression with panel corrected standard errors, natural logs of all variables and no lags

	WDI Under-Nutritic Infant	WDI Infant	WDI Under-five	DHS Neonatal	DHS Post Neonatal Infant	DHS Infant	DHS Child	DHS Under-five	DHS Adj Child	DHS Adj Infant
GDP Per Capita, PPP (current US\$)	-0.704***	-0.688***	-0.782***	-0.254***	-0.563***	-0.412***	-0.871***	-0.540***	-0.588***	-0.384***
Gini Index	0.637*	0.737***	0.845***	0.265	1.047***	0.691***	1.078**	0.822***	0.982**	0.395
Ratio of girls to boys in primary and secondary education	-1.245**	-0.948***	-1.174***	-1.111***	-1.186***	-1.156***	-2.328***	-1.532***	-1.424***	-1.237***
1989/1994Time dummy	11.40***	10.75***	0	9.316***	0	9.729***	16.14***	12.26***	0.0754	11.18***
1995/1999Time dummy	11.27***	10.74***	0.0133	9.357***	0.0974	9.805***	16.28***	12.35***	0.0561	11.13***
2000/2004Time dummy	11.38***	10.75***	0.0250	9.321***	0.0297	9.765***	16.36***	12.36***	0	11.00***
2004/2006Time dummy	0	0	0	0	0	0	0	0	0	0
Constant	0	0	12.41***	0	8.887***	0	0	0	11.44***	0
N	122	175	175	78	79	80	79	80	54	62
Adjusted R-Squared	0.683	0.929	0.923	0.909	0.783	0.833	0.867	0.918	0.922	0.926

Key * p<0.05 ** p<0.01 *** p<0.001"

Table 9: OLS Panel Data Regression with PCSE, Only Income Logged and No Lags

Panel OLS regression with panel corrected standard errors with only the log of income taken and no lag

	WDI Under-Nutrition	WDI Infant	WDI Under-five	DHS Neonatal	DHS Post Neonatal Infant	DHS Infant	DHS Child	DHS Under-five	DHS Adj Child	DHS Adj Infant
GDP Per Capita, PPP (current US\$)	-9.167***	-27.49***	-47.17***	-7.368***	-17.09***	-24.51***	-29.94***	-51.04***	-51.39***	-26.22***
Gini Index	0.0842	0.517***	0.804***	0.222	0.647***	0.854**	1.269***	2.260***	2.244***	0.870**
Ratio of girls to boys in primary and secondary education	-0.301***	-0.826***	-1.429***	-0.475***	-0.421***	-0.891***	-1.295***	-2.111***	-1.715***	-1.028***
1989/1994Time dummy	115.7***	327.0***	-8.591*	121.9***	168.8***	0	-12.54*	571.7***	2.758	12.04*
1995/1999Time dummy	114.0***	329.0***	-3.897	123.1***	173.1***	5.495	-4.972	584.2***	2.605	10.08*
2000/2004Time dummy	115.5***	330.3***	0	121.8***	172.6***	3.750	0	588.0***	0	0
2004/2006Time dummy	0	0	0	0	0	0	0	0	0	0
Constant	0	0	559.4***	0	0	291.3***	335.8***	0	536.7***	317.2***
N	122	175	175	78	79	80	79	80	54	62
Adjusted R-Squared	0.560	0.850	0.830	0.696	0.712	0.764	0.729	0.778	0.787	0.782

Key * p<0.05 ** p<0.01 *** p<0.001

Table 10: OLS Panel Data Regression with PCSE, All Variables Logged and Five Year Lag

Panel OLS regression with panel corrected standard errors and natural logs of all variables and a five year lag

	WDI Under-Nutrition	WDI Infant	WDI Under-five	DHS Neonatal	DHS Post Neonatal	DHS Infant	DHS Child	DHS Under-five	DHS Adj Child	DHS Adj Infant
GDP Per Capita, PPP (current US\$)	-0.720***	-0.721***	-0.815***	-0.268**	-0.639***	-0.439***	-0.934***	-0.582***	-0.612***	-0.316**
Gini Index	0.120	0.866***	0.931***	0.477	1.437***	0.755**	2.015***	1.169***	1.188**	0.367
Ratio of girls to boys in primary and secondary education	-0.555	-0.950***	-1.161***	-0.914*	-1.221**	-0.881**	-2.364***	-1.375***	-1.694***	-1.277**
1995/1999Time dummy	10.24***	-0.0195	12.15***	0.305*	0.0749	0.200	0.114	0.106	-0.229*	0
2000/2004Time dummy	10.23***	-0.0264	12.14***	0.349**	0.0246	0.203	0.251	0.158	0	0.00420
2004-2006Time dummy	10.14***	0	12.17***	0	0	0	0	0	0	0
Constant	0	10.42***	0	7.360***	7.964***	8.201***	12.95***	10.35***	12.03***	10.75***
N	75	175	175	49	50	51	50	50	27	35
Adjusted R-squared	0.702	0.927	0.932	0.858	0.674	0.910	0.734	0.876	0.855	0.808

Key

* p<0.05 ** p<0.01 *** p<0.001"

Table 11: OLS Panel Data Regression with PCSE, Only Income Logged and Five Year Lag

Panel OLS regression with panel corrected standard errors, only the natural log of income taken and five year lags of all the independent variables

	WDI Under-Nutrition	WDI Infant	WDI Under-five	DHS Neonatal	DHS Post Neonat	DHS Infant	DHS Child	DHS Under-five	DHS Adj Child	DHS Adj Infant
GDP Per Capita, PPP (current US\$)	-10.19***	-25.73***	-43.80***	-7.857**	-17.48***	-25.25***	-28.65***	-51.13***	-52.89***	-24.20***
Gini Index	-0.0838	0.642***	0.969***	0.400*	0.863***	1.286***	1.570***	2.648***	2.594***	0.829*
Ratio of girls to boys in primary and secondary education	-0.148*	-0.767***	-1.343***	-0.349**	-0.358**	-0.714***	-0.978***	-1.586***	-1.754***	-0.818***
1995/1999Time dummy	113.5***	296.4***	-9.826*	5.177	154.2***	3.457	266.8***	499.9***	-14.86*	0
2000/2004Time dummy	115.1***	299.3***	-4.116	6.710*	154.8***	5.577	271.9***	506.7***	0	-3.866
2004-2006Time dummy	111.4***	300.7***	0	0	155.1***	0	275.6***	504.5***	0	0
Constant	0	0	510.3***	98.51***	0	253.2***	0	0	529.0***	284.5***
N	75	175	175	49	50	51	50	50	27	35
Adjusted R-squared	0.587	0.816	0.792	0.627	0.677	0.720	0.727	0.766	0.789	0.631

Key:

* p<0.05 ** p<0.01 *** p<0.001"

Panel Data Model across Income Groups

So far we have seen that across all developing countries income, income inequality and women's power determine child survival. Yet these variables may not explain differences in child survival within a subset of lower-middle or upper-middle-income countries. This is because in these richer developing countries even children in poorer male dominated households may receive sufficient food and other basic goods. This means that variables that determine the amount of goods children consume such as income, income inequality and women's power may not be significant determinants of child survival in these richer developing countries. This thesis tests this proposition by rerunning the original panel data regression for under-five mortality for lower, lower-middle and upper-middle-income countries respectively³⁸.

The results from these regressions (see table 12) show that income, income inequality and women's power are significant determinants of under-five mortality in all income groups. The elasticities of income and income inequality are also fairly constant across income groups. There is no evidence that income and income inequality have a smaller or no impact on under-five mortality in richer developing countries. This suggests that even in lower-middle-income countries, children in poor, male dominated households may still receive limited food and other basic goods, reducing their chances of surviving.

The only unexpected result is that women's power increases under-five mortality in upper-middle-income countries. This last piece of evidence could be used to support the proposition that women's ability to control resources does not determine under-five mortality above a certain income threshold. However, if this were the case we would expect to see a systematic decrease in the elasticity between women's power and under-five mortality as countries become richer. As our results do not show this (the elasticity for women's power is higher for lower-middle-income than low-income countries) we do not find the result that women's power increases under-five mortality in upper-middle-income countries to be convincing.

³⁸ WB definitions of these income groups were used.

Table 12: OLS Panel Data Regression with Panel Corrected Standard Errors by Income Group

Panel OLS regression with panel corrected standard errors, natural logs of all variables with no lags, by income group (World Bank Definition)

	All Countries WDI Under five mortality	Low Income WDI Under five mortality	Lower Middle Income WDI Under five mortality	DHS DHS Under five mortality	Upper Middle Income WDI Under five mortality
GDP Per Capita, PPP (current US\$)	-0.782***	-0.642***	-0.405*	-0.540***	-0.728***
Gini Index	0.845***	0.818***	0.979***	0.822***	0.916***
Ratio of girls to boys in primary and secondary education	-1.174***	-1.077***	-2.383***	-1.532***	2.974*
1995/1999Time dummy	0	-0.149	14.31***	12.26***	-7.235
2000/2004Time dummy	0.0133	-0.0684	14.35***	12.35***	-7.820
2004-2006Time dummy	0.0250	0	14.28***	12.36***	-8.040
Constant	12.41***	11.14***	0	0	0
N	175	83	67	80	24
Adjusted R-Squared	0.923	0.916	0.919	0.918	0.928

Key * $p < 0.05$ ** $p < 0.01$ *** $p < 0.001$ "

Is it Women's Power or their Education that Affects Mortality?

Some earlier studies found that women's education reduced child mortality. We have, however, argued that it is women's power, mainly through their ability to distribute household resources to children, which determines child mortality. This raises the possibility that earlier work found women's education significant because it acts as a proxy for their power. Yet it could also be this thesis which is wrong. It could be that our measure of women's power (the ratio of girls and boys in primary and secondary education) is highly correlated with, and acting as a proxy for, women's education.

This section investigates the relationship between women's education, women's power and child mortality. We use two measures of women's education. The first is female persistence to grade five of schooling as a percentage of their cohort. This shows the number of girls who are still attending school at age five divided by the total number of girls eligible to attend school. This percentage will be lower in societies where many girls do not attend school and where female students drop out before grade five. Our second measure is the female primary completion rate as a percentage of the relevant age group. This shows the number of girls who complete secondary school divided by the total number that were eligible to attend school.

Table 13 shows the results for different models that include different measures of women's power and education. The first column shows, using WDI data, our original model where under-five mortality is the dependent variable and income, income inequality and women's power are independent variables. All the independent variables are significant at the 1% level and the adjusted R-Squared is 0.923. The second model (column 2) replaces women's power with our first measure of their education (female persistence to grade five of schooling as a percentage of their cohort). In this model the coefficient on female education is negative but insignificant. This suggests that while women's power is a significant determinant of under-five mortality their education may not be.

The model in column 3 includes under-five mortality (WDI) as the dependent variable and income, income inequality and women's education (measured by the primary completion rate as a percentage of their cohort) as independent variables. The coefficient on primary education is negative and significant at the 1% level. This result shows how earlier multivariate studies could have concluded that it is women's education that reduces under-five mortality. The coefficient on women's education is, however, much smaller than the coefficient on women's power was in our original model. This suggests the impact of women's power on under-five mortality is larger than that of women's education.

The models in columns 4 and 5 further investigate the relationship between under-five mortality and women's power and education. The model in column 4 includes under-five mortality (WDI) as the dependent variable and income, income inequality, women's power and female education (as measured by their persistence to grade five) as independent variables. Income, income inequality and women's power are all highly significant but female education is insignificant. The model in column 5 has an identical specification but female education is measured by the female primary completion rate. Income, income inequality and women's power are significant and female education is again insignificant.

Our results are best interpreted with reference to our analytical framework. This showed that female education reduces child mortality directly through its impact on the personal illness control of children (including health interventions). And that female education can also indirectly reduce child mortality by increasing individual women's power. More powerful women may have lower fertility and better birth spacing, demand and receive better medical care and ensure that children receive a larger share of household consumption.

When women's education (as measured by the primary completion rate) is included in our model it is significant. This could be because the total impact of this variable, including its impact on mortality through mother's power, is significant. The larger coefficient on women's power when only it is included supports this result and suggests that women's power is not entirely determined by female education. The insignificance of female education when women's power is also included suggests that its partial effect is insignificant. So in conclusion our results suggest that female education can significantly reduce under-five mortality by increasing women's power but not directly through better knowledge of health interventions. Women's power is, however, determined by many variables and therefore when trying to explain under-five mortality across countries and time it is a more powerful independent variable than women's education.

The conclusion that female education cannot directly reduce under-five mortality should be interpreted cautiously. Our econometric models provide average results across all countries. Just because female education did not, once mother's power was controlled for, on average across all countries significantly reduce under-five mortality through knowledge of specific health interventions does not mean that it cannot do so in countries which have especially effective female education programmes. Our measure of female education is a measure of the number of girls that attended school and not the quality of the education they receive. Some countries may have particularly high quality education systems or curriculums which emphasize knowledge of basic health interventions and in such countries female education may directly reduce under-five mortality through knowledge of health interventions.

How does Income Inequality Affect Mortality?

Our analytical framework showed that income inequality increased child mortality because there are diminishing marginal returns (aggregation hypothesis) between child health and families' income. Increasing a poor family's income from \$100 to \$200 is more likely to save a child's life than increasing a rich family's income from \$10000 to \$10100. The evidence appears to support this hypothesis; with increased income inequality significantly worsening child survival across a range of models. Yet these results are also consistent with two alternative explanations. First, income inequality could be acting as a proxy for the power of different groups to control government health expenditure. Second, it could be that the rich being rich causes, through some psychosocial mechanisms or a reduction in social capital, increased mortality among the poor. How can we test which of these causal pathways is correct?

We answer this question using the following logic: if we hold the actual income of the poorest groups in society constant then a significant negative coefficient on the share of income held by the rich provides evidence that income inequality does not work solely through the diminishing marginal returns to income pathway. Yet if the rich getting richer, when the poor are not getting poorer, reduces or does not increase child mortality, then in our

original model, inequality must have affected child mortality solely through the diminishing marginal returns to income pathway.

The models in table 14 test this logic³⁹. The first model (column 1) includes women's power and the real income of the poorest and richest 10% of society as determinants of under-five mortality (WDI). The income of the rich has a significant negative coefficient. That is the rich getting richer, when the poor are not getting poorer, reduces child mortality. This is confirmed by the second model (column 2) which includes the real income of the poorest and richest 20%. Again, the coefficient on the real income of the rich is negative and significant.

However, the third and fourth models, which include women's power, income and the income of the richest and poorest 10% (column 3) or 20% (column 4) of society, both show a positive significant coefficient on rich people's share of income. That is, when a country's income and the real income of the poor are held constant the rich getting richer increases mortality. This result could be interpreted as supporting the psychosocial hypothesis; the poor's health suffers when the rich get richer. But it is also consistent with the diminishing marginal returns to income hypothesis because the decrease in income suffered by those not in the poorest 10% or 20% of society, when the rich get richer and average income remains the same, could still lead to increased under-five mortality. Model 5 (column 5) investigates this further. It demonstrates that holding a country's average income, the income of the poorest 20% and poorest 20 to 40% of society constant, that the income of the richest 20% has no significant impact on under-five mortality. It is thus unlikely that income inequality affects under-five mortality through the psychosocial mechanism, a reduction in social capital or acts as a proxy for socioeconomic groups' ability to control government policy. This is confirmed by model 7 column 7. It shows that holding the income of the poorest 20% and poorest 20% to 40% constant, the Gini index does not significantly affect mortality. So the Gini index cannot be acting as a proxy for the overall power of the rich compared to the rest of society. Rather in our original model income inequality (as measured by the Gini index) was significant because in more unequal societies the poor have lower average incomes and the rich higher incomes. This increases mortality because there are diminishing marginal returns between family income and under-five mortality.

In conclusion, income inequality increases under-five mortality across countries because there are diminishing marginal returns between family income and under-five mortality. It does not affect under-five mortality through the psychosocial mechanism or reduced social capital and it is not acting as a proxy for the power of different socioeconomic groups.

³⁹ Waldmann (1992) uses a similar logic.

Table 13: Is it Women's Power or Education that Affects Mortality? (All Variables are Natural Logs with no Lag)

Column	1	2	3	4	5
	Original Model WDI Under-five mortality	Female Education 1 WDI Under-five mortality	Female Education 2 WDI Under-five mortality	Power and Education 1 WDI Under-five mortality	Power and Education 2 WDI Under-five mortality
GDP Per Capita, PPP (current US\$)	-0.782***	-0.854***	-0.767***	-0.738***	-0.763***
Gini Index	0.845***	0.323	0.558***	0.562**	0.787***
Ratio of girls to boys in primary and secondary education	-1.174***			-1.213***	-0.860**
1995/1999Time dummy	0.0133	0.0640	-0.0150	0.115	0.0391
2000/2004Time dummy	0.0250	0.0270	-0.00425	0.0844	0.0577
Female persistence to grade five (% of their cohort)		█-0.186		█-0.146	
Primary completion rate female percent of relevant age group			█-0.320***		-0.124
Constant	12.41***	10.48***	9.412***	13.93***	11.54***
N	175	116	202	102	158
Adjusted R-Squared	0.923	0.954	█0.937	0.954	0.930
Key	=** p<0.05	** p<0.01	*** p<0.001"		

Table 14: How Does Income Inequality Affect Mortality? (All Variables are Natural Logs with No Lag)

Column	1	2	3	4	5	6	7	8
	WDI Under-five mortality	WDI Under-five mortality	WDI Under-five mortality	WDI Under-five mortality	WDI Under-five mortality	WDI Under-five mortality	WDI Under-five mortality	WDI Under-five mortality
Ratio of girls to boys in primary and secondary education	-1.378***	-1.299***	-1.124***	-1.123***	-1.123***	-1.464***	-1.148***	-1.133***
Average real income of the poorest 10 % of society in GDP Per Capita PPP	-0.429***		█0.0524					
Average real income of the richest 10 % of society in GDP per Capita PPP	-0.339***		█0.966***					
1995/1999Time dummy	0.00319	0.00918	0.0235	0.0271	0.0444	0.0124	0.0532	0.0416
2000/2004Time dummy	0.0260	0.0423	0.0279	0.0210	0.0467	0.0497	0.0639	0.0481
Average real income of the poorest 20 % of society in GDP Per Capita PPP		█-0.514***		0.169	0.375	-0.755***	0.492*	0.474*
Average real income of the highest 20 % of society in GDP Per Capita PPP		█-0.274***		1.613***	0.870			
GDP Per Capita PPP (for country)			█-1.796***	-2.559***	-1.270			-0.322
Average real income of the poorest 20 to 40 % the society in GDP Per Capita in PPP					█-0.757		-1.274***	-0.932*
Gini Index						-0.471**	0.235	0.610
Constant	16.09***	15.73***	14.22***	14.07***	14.47***	17.41***	14.23***	13.00***
N	175	175	175	175	175	175	175	175
Adjusted R-squared	0.933	0.928	0.920	0.925	0.929	0.923	█0.930	0.923
Key		** p<0.01	*** p<0.001"					

Does Health Expenditure Reduce Under-five Mortality?

Government health expenditure (table 15 column 2) does reduce under-five mortality but its impact is small. The coefficient on the log of government health expenditure is just -0.125; meaning that a 1% increase in government expenditure on health per capita causes a 0.125% reduction in mortality. In addition the inclusion of government health expenditure per capita only increases the adjusted R-Squared from 0.819 to 0.829. Income, income inequality and women's power continue to be significant and of the expected sign in all the models discussed in this section.

The coefficient of -0.6 on income in this model shows the partial elasticity between income and under-five mortality which does not operate through increased government health expenditure. The large difference between this coefficient and the coefficient on government health spending suggests that policies which directly increase people's income will be more effective in reducing under-five mortality than government health expenditure. This result is broadly consistent with the literature. Filmer and Pritchett (1999) find that public expenditure on health is a significant determinant of, but that it explains little of the variation in, under-five mortality. The incremental R-squared in their model due to including public health expenditure is just 0.0015. These authors also find that including public expenditure on health only results in a decrease in the elasticity between income (GDP per capita) and under-five mortality from -0.645 to -0.596. This suggests that income reduces under-five mortality mainly through family income and not through increased spending on healthcare.

We also estimated (table 15 column 3) the impact of total health expenditures on under-five mortality. This significantly reduced mortality, with a 1% increase in total health expenditure leading to 0.2% decrease in mortality. The larger coefficient on total health expenditure (-0.199) compared to public health expenditure (-0.125) suggests that private health spending has a significant but small impact on under-five mortality. The continuing significance of income when total health expenditure is controlled for suggests that increases in income, even when they are not spent on healthcare, have the ability to decrease under-five mortality.

The above results implicitly assume that public and total health expenditures' impact is the same across all countries. Yet this is implausible. The reality is that for a variety of reasons the effectiveness of public health expenditure in reducing under-five mortality is likely to vary across countries. Our analytical framework argued that an important determinant of the effectiveness of public expenditure was state capacity. Countries with high state capacity should have more effective health expenditures. It may also be the case that high capacity governments are able to better regulate the private health sector; increasing the effectiveness of total health expenditures.

This chapter aims to explicitly test these hypotheses. A state capacity variable was created based on the average of three CPIA measures, namely: public sector management and institutions; quality of budgetary institutions and financial management and the quality of public administration. These measures were chosen because they are directly relevant to the efficacy of public spending. These terms were interacted with public health expenditure to determine if health expenditure had a greater impact on under-five mortality in countries with high capacity states. The interaction term was, however, insignificant (table 15 column 4). As was the interaction term based on government capacity and total health spending (table 15

column 5). Other measures of state capacity based on CPIA indexes were also constructed, but these also showed that state capacity does not affect public expenditures' impact on child health⁴⁰.

These results should be interpreted with caution. The CPIA measure has only been collected for a small number of developing countries for a few years. The regressions that include this variable, thus, only contained 38 observations and consequently they are unlikely to find significant results. The CPIA index also has very similar values for the majority of developing countries in our regression, which perhaps suggests that it cannot accurately determine small but significant differences in capacity between different developing countries' governments.

A further theoretical problem with this model is that it only allows the effectiveness of public expenditure to vary with government capacity. There are likely to be capacity constraints on the effectiveness of health expenditures but political constraints may be equally important. In many developing countries powerful socioeconomic groups effectively control the state and use government expenditure for purposes of political patronage⁴¹. In such countries government health expenditure is unlikely to efficiently reduce under-five mortality. On the other hand, there may be developing countries where poorer socioeconomic groups, who would benefit most from public expenditure targeted at reducing under-five mortality, can influence policy, or where for other political reasons, governments are keen to effectively reduce under-five mortality; in these countries government expenditure may be effective.

This chapter needs to find a variable that broadly measures government capacity and political commitment to reduce under-five mortality. The UK Department for International Development's (DFID) measure of whether or not a state is fragile arguably meets this criterion. Its definition of fragile states is one where the government cannot or will not deliver core functions to the majority of its people, including the poor (DFID 2005)⁴².

So is government health expenditure more effective in non-fragile than fragile states? Our next model investigates this relationship by running two separate regressions, one for fragile states and one for non-fragile states. For non-fragile states public expenditure does significantly reduce under-five mortality (see table 16 column 1), but for fragile states its coefficient is positive and insignificant (see table 16 column 2). The second fragile state regression contains just 14 observations; it is thus unsurprising that the positive coefficient on government health expenditure is insignificant. These two regressions provide some evidence that government health expenditure is less effective in fragile states.

⁴⁰ Results not reported here.

⁴¹ See: Peters et al (1999) for a discussion of how politics may undermine the effectiveness of health spending. See Kohli (2004) for a discussion of how patronage based politics may weaken the state.

⁴² We are not arguing in this thesis that whether a state is fragile or not is an adequate measure of state power or political commitment. Indeed throughout this thesis we argue that analyzing the impact of these variables on the efficacy of social policy in reducing under-five mortality requires a detailed case study of a particular country. Rather we are arguing that if an attempt is going to be made to include such variables in a regression analysis, DFID's definition of whether or not a state is fragile is better than most other measures available because it, or at least makes an attempt to, account for political variables.

Our final two models examine the impact of total health expenditure in fragile and non-fragile states. It finds that total health expenditure significantly⁴³ reduces mortality in non-fragile states (table 16 column 3), but is insignificant in fragile states (table 16 column 4). This result demonstrates that government capacity and countries' political commitment may affect child health through both private and public healthcare.

⁴³ At the 10% level of significance.

Table 15: Health Spending (All Variables are Natural Logs with No Lag)

Column	1	2	3	4	5
	WDI	WDI Under-five mortality	WDI Under-five mortality	WDI Under-five mortality	WDI Under-five mortality
GDP Per Capita, PPP (current US\$)	-0.780***	-0.600***	-0.747***	-0.680***	-0.841***
Gini Index	0.845***	0.980***	0.912***	1.118**	1.136**
Ratio of girls to boys in primary and secondary education	-2.129***	-1.874***	-1.854***	-1.706**	-1.638**
Government health expenditure per capita in USD (GHEP)		-0.125*			
Total health expenditure per capita in USD (THEP)			-0.199*		
Government capacity * GHEP				-0.0343	
Government capacity * THEP					-0.0715
Constant	16.72***	14.23***	15.14***	13.49***	14.05***
Sample size	75	75	76	38	38
Adjusted R-squared	0.819	0.829	0.827	0.713	0.717
Key	="** p<0.05 ** p<0.01 *** p<0.001"				

Table 16: Health Spending in Fragile and Non-fragile States (All Variables are Natural Logs with no Lag)

Column	1	2	3	4
	WDI Under-five mortality Not Fragile	WDI Under-five mortality Fragile	WDI Under-five mortality Not Fragile	WDI Under-five mortality Fragile
GDP Per Capita, PPP (current US\$)	-0.612***	-0.253	-0.776***	-0.292
Gini Index	0.972***	0.759	0.906***	0.955
Ratio of girls to boys in primary and secondary education	-1.579**	-2.659**	-1.644**	-2.457**
Government health expenditure per capita in USD (GHEP)	-0.137*	0.0923		
Total health expenditure per capita in USD (THEP)			-0.188	-0.0632
Constant	13.11***	15.24**	14.48***	14.25**
Observations	60	14	62	14
Adjusted R-squared	0.819	0.691	0.812	0.683
Key	="** p<0.05 ** p<0.01 *** p<0.001"			

Note THEP Not fragile is significant at 10 % level

Conclusion

This section empirically tested our analytical framework. Our econometric models provide striking evidence that income, income inequality and women's power determine child survival and under-nutrition. These results were confirmed using different estimation techniques, functional forms, lag structures and measures of child survival. So our results are robust and did not occur by chance. What do our results tell us about how countries can reduce child mortality?

Whether or not governments can reduce child mortality partly depends on the extent to which they can control income, income inequality and women's power. Can governments easily change these variables or are they structural factors beyond, at least in the short term, their control? The extent to which economic growth is structural is a complex question and one beyond our remit. Here we simply note that when countries committed themselves to MDG4 and reducing child mortality they were not principally committing themselves to increased economic growth; and that if we were to regard economic growth as non-structural our policy advice to developing countries would be: "grow faster" — a statement as useless as it is simple.

Whether income inequality is a structural factor is also complex and debatable. Our econometric models demonstrate that increased income inequality increases child mortality mainly through its association with family income. This, coupled with the high partial elasticity of income, not operating through government health expenditure, demonstrates that redistribution policies that improve poor families' incomes may be effective in improving child survival. Yet we should be cautious about concluding that all governments at all times and in all places should start redistributing income. For the coefficient on income inequality represents actual income redistribution: not the effectiveness of government policies that redistribute income. Redistribution policies will be plagued by the same capacity, corruption and political constraints that constrain the effectiveness of public health expenditure. So the relative efficiency of income redistribution and health programmes needs to be analyzed before we can reach a conclusion regarding which is most cost effective for reducing child mortality.

Women's power also improves child survival. The obvious conclusion is that developing countries' governments should implement policies that increase women's status and standing in society. Yet, the extent to which governments can influence such cultural considerations in the short and medium term is debatable. It may, therefore, be the case that women's power is best regarded as a structural variable that cannot be easily changed by governments and that it is best to devise goals and policies that take this into account.

Our results also showed, that at least according to our econometric evidence, women's education does not significantly reduce child health through knowledge of specific health interventions, but that studies could find it significant because it acts as a proxy for, or partially determines, women's power within society. This result means that the effectiveness of women's education programmes may be less than previously thought. And that only those programmes that broadly increase women's power and status within society and the household are likely to improve child health. Programmes that only increase a mother's knowledge of hygiene and other medical interventions are unlikely to be effective.

In summary, income, income inequality and women's power are at best only partly controlled by government. The ability of governments to improve child survival through policies in these areas is likely to be limited in the short term.

Governments could, however, improve child survival in the short term by implementing effective health policies. Our econometric model found, similar to other studies (Filmer and Pritchett 1999), that government expenditure on health reduced mortality but not by very much. The obvious, yet incorrect, conclusion is that governments cannot, and thus should not try, to improve child health. Such a conclusion is wrong because our model shows that across all countries government health expenditures' impact on child mortality is actually low: not that it does not have the potential to reduce child mortality. Epidemiological (Jones et al 2003) studies and studies of health interventions (Bryce et al 2003) show that government policies can potentially reduce mortality.

The conundrum is why government expenditure can potentially, but on average actually is not, effectively improving child health. We argued that state capacity and politics are the most likely explanations. However, our econometric evidence in support of this hypothesis is at best weak. The main technical explanation for this result is that limited data on state capacity is available. The deeper theoretical explanation is that the interaction between politics, state capacity and public expenditures' effectiveness is inherently complex and that countries' politics are difficult to measure. Politics and state capacity do not lend themselves to the kind of reductionist criteria that is needed to include variables in a regression analysis.

Governments can also improve child health through policies other than healthcare. Our analytical framework, for example, shows that water supply and sanitation can improve rates of child survival. Yet studying the effectiveness of these interventions through our panel data econometric model would be problematic because the effectiveness of such social policies are also likely to be determined by state capacity and politics – which are inherently difficult to measure.

An alternative methodology is to identify countries that have high under-five mortality for their structural factors (income, income inequality and women's power). Table 17 shows, using the results from our cross-country regression, those countries which have high and low under-five mortality for their income, income inequality and women's power. The first seven outliers from the WDI dataset are shown, but due to a smaller sample size only the first three countries from the DHS dataset are shown.

Table 17: Outlying Countries

Countries that have lower than predicted under-five mortality. Largest outlier (e.g. much lower than predicted) first				
	Holding Income Constant		Holding income, income inequality and women's power constant	
	DHS Data	WDI Data	DHS Data	WDI Data
1	Moldova	Moldova	Moldova	Moldova
2	Vietnam	Croatia	Vietnam	Chile
3	Uzbekistan	Jamaica	Colombia	Jamaica
4		Dominica		Yemen, Rep.
5		Belarus		Costa Rica
6		Sri Lanka		Vietnam
7		Vietnam		Malaysia
Countries that have higher than predicted under-five mortality. Largest outlier (e.g. much higher than predicted) first				
	Holding Income Constant		Holding income, income inequality and women's power constant	
	DHS Data	WDI Data	DHS Data	WDI Data
1	Guatemala	Equatorial Guinea	Guatemala	South Africa
2	Gabon	Gabon	Bangladesh	Kazakhstan
3	Cote d'Ivoire	South Africa	Kazakhstan	Guyana
4		Swaziland		Algeria
5		Guinea		Swaziland
6		Botswana		Mauritania
7		Namibia		Bangladesh

Our argument is that countries that have low under-five mortality for their structural factors will have effective social policies. And that a country's state capacity and politics will determine the efficacy of its social policies. This analysis selects Vietnam as a country with effective social policies because it has low under-five mortality for both its income and its structural factors in both the DHS and WDI datasets. Vietnam was also consistently an outlier when we used different measures of child survival, functional forms and lag structures⁴⁴.

There were three further reasons for selecting Vietnam as a case study. First, it is one of the few countries currently on course to achieve MDG4. This in itself makes it an interesting case study. This is discussed in more detail in the next chapter. Second, two recent DHS (1997 and 2002) have been carried out in Vietnam and are publically available. This means that accurate data on child mortality, child nutrition, fertility, the proximate determinants of fertility, maternal and childcare, infant feeding and availability of healthcare exist for two different points in time. This data was used in our case study analysis and was not always available for other countries. The four rounds of the VLSS also provide detailed data on poverty, income and expenditure. Third, prior to writing this thesis, I had a better knowledge of Vietnam's institutions and culture than those of the other outlying countries, having previously worked in Vietnam.

The main potential criticism with our method of selecting Vietnam, because it is an outlier, is that the unexplained variation in the regression model may not reflect the complex interactions between government capacity, politics and the effectiveness of public expenditure, but may instead simply be random variation. Here there are two rebuttals. First, this argument is applicable to any analysis which selects cases based on them being outliers. Earlier work that identified Sri Lanka as having high human development for its income may simply have been identifying random variation (Isenman 1980 and Sen 1981). Second, we regard Vietnam's status as an outlier as providing a strong indication, but not absolute proof,

⁴⁴ Results not reported.

that it has effective social policies. We are saying that our econometric analysis leads us to believe that Vietnam has effective social policy and does other evidence support this? Do academic and donor evaluations conclude that Vietnam's social policies have improved child health? Do other academic studies conclude that Vietnam has effective social policies? And are the underlying factors behind this effective social policy, high state capacity and amenable politics?

To conclude, this chapter provided robust econometric evidence demonstrating that as predicted by our analytical framework: a) income, income inequality and women's power determine child survival and under-nutrition across countries and time; b) income inequality affects mortality through its association with family income; and c) it is women's power not their level of education that affects mortality. We also selected a country – Vietnam – which has low child mortality for its structural factors. Vietnam's outlying status should be explained by the effectiveness of its social policies, which may in turn be explained by the capacity of its state and its politics.

Chapter 5: Child Mortality and Social Policy in Vietnam

Introduction

This chapter concentrates on examining whether social policies can explain why Vietnam has low child mortality, given its structural factors. The fourth and longest section of this chapter describes, and evaluates the impact on child mortality of each social policy.

Prior to undertaking a detailed review of social policies in Vietnam, this chapter contains three shorter sections. The first discusses the availability of data on child mortality and its determinants. The second reviews the existing literature on the determinants of child mortality. The third discusses the distribution of child mortality by family income, region and ethnicity and whether the distribution of child mortality in Vietnam can explain why it has low child mortality for its structural factors. This section also discusses the epidemiology of child mortality.

The final section of this chapter briefly discusses in a few pages the state institutions and distribution of power between different groups which underpinned the implementation of social policies in Vietnam. The historical process that led to the development of effective state institutions and a narrow concentration of political power in Vietnam is also briefly discussed.

5.1 Review of Available Data Sources

Analysis of the trends in and the distribution of child mortality are dependent on the available data. Table 18 lists the main sources of nationally representative household survey data on child mortality and its determinants in Vietnam.

This chapter uses data from all of the household surveys listed in table 18. The majority of this data is, however, drawn from the 1992/1993 VLSS, 1997/1998 VLSS, 1997 DHS, and 2002 DHS and 2006 MICS. The reasons for concentrating on these five core surveys are further discussed below.

Together these five core surveys include relevant data from the 1990s until 2006. This is the most relevant period for our analysis because our econometric models showed that Vietnam had low child mortality for its structural factors using dependent variable data until 2006.

These five surveys include data on child mortality and many of its likely determinants. All five surveys include data on child mortality, anthropometric indicators, fertility, family planning, parents' educational attainment and water and sanitation. This means that the statistical relationship between child mortality and many of its likely determinants can be examined at the household level using these datasets.

The 1997 DHS, 2002 DHS and 2006 MICS also include data on child morbidity. Such data was not collected in the VLSS 1992/1993 or VLSS 1997/1998. Both these VLSS did,

however, collect detailed data on household expenditure and income and spending on healthcare. Neither household income nor expenditure data were collected in the 1997 DHS, 2002 DHS or 2006 MICS. These three surveys did, however, collect data on household assets. Using VLSS data in addition to DHS and 2006 MICS data allows us to discuss the distribution of child mortality by income and the relationship between morbidity and mortality.

A national health survey was also conducted in Vietnam in 2001/2002. We do not make extensive use of the statistics from this survey because it only contains data on child deaths for the three years immediately preceding the survey and in addition it does not contain data on families' assets, expenditure or income. This means that DHS and VLSS data are more relevant for much of the analysis undertaken in this chapter.

VLSS were also carried out in 2002, 2004 and 2006. Statistics from these surveys are not used as extensively as data from our five core surveys because these later VLSS did not collect child mortality or anthropometric data. In addition, the 2002 survey did not collect any data on morbidity, fertility or family planning. And the 2004 survey did not collect any data on fertility or family planning. These surveys cannot be used to directly assess the relationship between household income and child mortality across families or regions. These surveys can, however, be used to show national or regional trends in likely determinants of child mortality, such as poverty, and this can be compared to child mortality data from other sources. We make use of poverty data from the 2002, 2004 and 2006 VLSS in this chapter.

This chapter also uses data taken from international databases such as WDI. These databases provide access to data on child mortality and its determinants for many developing countries. As discussed earlier, this data is sometimes taken from national statistics offices and can be inaccurate as many countries cannot accurately record all child births and deaths. Other data points are based on interpolations or household surveys and may not be strictly comparable across time or countries. The original source of child mortality data in these datasets is often difficult to trace. We therefore only use data from international datasets for one of three reasons. First, when data from household surveys in Vietnam for a particular year was not available. Second, to cross check our findings from an analysis of household survey data. We also use data from international databases to compare Vietnam's social policies to those of other developing countries.

We do not proceed by describing the results of each household survey in turn. Discussing trends in child mortality, its distribution and determinants based on VLSS data and then mimicking this discussion using DHS data would be highly repetitive. Rather we use the most relevant and accurate data available when discussing trends in child mortality over time and its distribution by family income, region and ethnicity.

Our discussion of child mortality by ethnicity uses data from the 1999 census because the sample size of most household surveys is insufficient to calculate child mortality rates for individual ethnic groups. Household surveys are, however, used to calculate child mortality rates for the ethnic minorities (which includes many different ethnic groups) and the ethnic majority (which includes the Viet and Hoa groups).

To summarize, the first three sections of this chapter mainly use data from the VLSS, DHS and 2006 MICS. However, some data from the 1999 census and international datasets are also used.

Table 18: Relevant Household Survey Data in Vietnam from 1992 to 2006

Household survey name and year	Sample size / number of households	Contains child mortality data?	Contains data on family income, expenditure or assets?	Contains data on the proximate determinants of child mortality? ⁴⁵	Contains data on preventative care and family planning?	Contains data on curative care?	Contains data on health expenditure?	Contains data on other relevant social policies?	Contains data on water and sanitation
1992/1993 Vietnam Living Standards Survey	4,800	Yes (from complete birth history)	household income and expenditure data collected	Anthropometric, fertility and breastfeeding data collected	Child immunization and family planning data collected	Data on distance to nearest health facility and type of healthcare sought collected	Yes	Education attainment data collected	Water and sanitation data collected
1997/1998 Vietnam Living Standards Survey	6,000 (including 4800 households from VLSS 1992/1993)	Yes (from complete birth history)	household income and expenditure data collected	Anthropometric, fertility and breastfeeding data collected	Child immunization and family planning data collected	Data on distance to nearest health facility, type of healthcare sought and quality of commune health facilities collected	Yes	Educational attainment and distance to educational facility data collected	Water and sanitation data collected
1997 DHS	5,500 (ever married women)	Yes (from complete birth history)	Household assets but not income or expenditure data collected	Anthropometric, fertility, breastfeeding and child morbidity (for diarrhoea, cough, fever) data collected	Child immunization and family planning data collected	Data on treatment of childhood illness and availability of healthcare collected.	No	Mother's educational attainment data collected	Water and sanitation data collected
2000 MICS	7,628 (households)	Yes from number of children ever born and number of surviving children	Household assets but not income or expenditure data collected	Anthropometric data for children, data on morbidity but only for diarrhoea, cough and fever, data on fertility and breastfeeding	Child immunization and family planning data collected	Data on treatment of childhood illnesses	No	Educational attainment data collected	Water and sanitation data collected
2001/2002 Vietnam National Health Survey	36,000 households	Only contains data on child deaths in last three years	No expenditure or asset index	Anthropometric data for children Morbidity data collected	Child vaccination data, family planning data collected	Data on treatment of childhood illness and data on availability of healthcare	No	No	No

⁴⁵ The data on morbidity collected by surveys are for all household members (unless otherwise stated) for two or four weeks preceding the survey. This means that data on child morbidity are for children who were often alive at the time of the survey and not normally for children who were reported to have died. The data on morbidity does not then provide the medical cause of death for each recorded child death. These morbidity data are, however, still useful as it is possible to calculate statistics comparing incidences of recent child morbidity and child mortality within families (albeit not for the same child) and comparing average child mortality and morbidity across families.

Household survey name and year	Sample size / number of households	Contains child mortality data?	Contains data on family income, expenditure or assets?	Contains data on the proximate determinants of child mortality? ⁴⁵	Contains data on preventative care and family planning?	Contains data on curative care?	Contains data on health expenditure?	Contains data on other relevant social policies?	Contains data on water and sanitation
2002 DHS	5,706 (ever married women)	Yes (from complete birth history)	Household assets, but not income or expenditure, collected	Anthropometric, fertility, breastfeeding and child morbidity (for diarrhoea, cough, fever) data collected	Child immunization and family planning data collected	Data on treatment of childhood illness and availability of healthcare collected	No	Mother's educational attainment data collected	Water and sanitation data collected
2002 Vietnam Household Living Standards Survey	30,000	No	Household income and expenditure data collected	No	No	Data on visits to health provider in last 12 months collected	Yes	Educational attainment data collected	Water and sanitation data collected
2002 World Health Survey	4,173	No	Household income and expenditure data collected	Data on common childhood illness collected	Data on vaccination, family planning, antenatal care collected	Data on visits to and quality of health providers collected	Yes	Educational attainment data collected	Water and sanitation data collected
2004 Vietnam Household Living Standards Survey	30,000	No	Household income and expenditure data collected	Child morbidity data collected	No	Data on visits to health provider in last 12 months collected	Yes	Educational attainment data collected	Water and sanitation data collected
2006 MICS	8,355	Yes, number of children ever born and number of surviving children	Household assets but not income or expenditure data collected	anthropometric data for children data on morbidity but only for diarrhoea, cough and fever, data on fertility and breastfeeding	Child vaccination data Family planning data	Data on treatment of childhood illnesses	No	Educational attainment	Data Water and Sanitation facilities
2006 Vietnam Household Living Standards Survey	45,945	No	Household income and expenditure data collected	Morbidity data collected Fertility data collected Anthropometric data for children	Contains family planning data	Data on visits to health provider in last 12 months	Health expenditure data collected	Educational attainment	Water and sanitation facilities

5.2 Studies Examining the Determinants of Child Mortality

This section reviews studies which discuss the determinants of child mortality in Vietnam. It reviews studies by Swenson et al (1995), Hoa et al (1997), WB (2002), ADB (2002), WHO (2007) and UNICEF (2010). These studies are discussed in order of the date by which they were published. Studies that examine the distribution of child mortality by region, ethnicity and income are discussed in the third section of this chapter. The WB (2002) and UNICEF (2010) studies also analyze the distribution of child mortality by income and their results in this area are further discussed in the third section of this chapter.

This literature review has two aims. The first is to examine whether the determinants included in our analytical framework can explain variation in child mortality in Vietnam. Do family income, mother's power and education, health outputs and water and sanitation explain differences in child mortality? The second aim of this review is to identify mechanisms through which social policies could have reduced child mortality in Vietnam and thus explain why it has low child mortality for its structural factors.

Swenson et al (1995) examine the socioeconomic determinants of child mortality in Vietnam. Their analysis uses data from the 1988 DHS and included children born between 1983 and 1988. A hazard model was used and independent variables were included to test for birth order, family income, commune size, mother's education, mother's age and province. Family income and birth order were significant; with children of birth order five and above facing a significantly increased risk of mortality. Commune size was also significant (rural children in communes of less than 10,000 faced a higher risk of mortality) and the province children lived in was of borderline significance. Mother's age and education were not significant determinants of mortality.

Hoa et al (1997) analyze the relationship between socioeconomic factors, child morbidity and child mortality. Their sample consists of 1,132 mothers in rural areas from the Red River Delta region with children less than five years of age. Mothers were interviewed about their reproductive history, child morbidity and child survival. Causes of child death were established using the verbal autopsy technique.

This study found high morbidity among children. Specifically 46% of children suffered from acute respiratory infection (ARI). Poor families were found to have higher rates of ARI among their children. Mortality among children was low. Only 81 children died below the age of five (out of 2,413 children) and two thirds of deaths occurred during the first year of life. These deaths were mainly related to prematurity, asphyxia or tetanus.

Cox proportional hazard analysis was undertaken to determine the relationship between socioeconomic factors, morbidity and mortality. Higher birth order and young age of the mother were both found to significantly increase the risk of child mortality. The education of the mother and her occupation were not found to be significantly related to child mortality. Family income was also found to be an insignificant determinant of child mortality. The insignificance of mother's education and family income is surprising as it contradicts much earlier work. This result may be explained by the area being relatively homogenous in terms of socioeconomic conditions and the sample size being too small to detect the impact of small differences in the independent variables on mortality. A further limitation of the study is that

the sample was drawn entirely from the Red River Delta region which may not be representative of Vietnam as a whole.

This study found two pieces of evidence which support the conclusion that there is good healthcare in Vietnam. First, child mortality was low despite high morbidity; possibly indicating that healthcare interventions were effective. Second, two thirds of children who were sick with ARI had been treated with antibiotics.

The WB (2002) seeks to explain the causes of child mortality in Vietnam. This study uses a reduced form demand equation to model child survival. This links child survival to household resources and variables which affect the shadow price of child survival. Variables that lower the shadow price of child survival such as the availability and quality of medical services, ought to increase households' "demand" for child survival. In contrast, variables such as poor local sanitary conditions which raise the shadow price of child survival would be expected to be negatively associated with child survival in the survival demand function.

In such a model, increases in the mean survival time can be due to either movements along the demand equation or shifts in the demand equation. Rising incomes can cause shifts along the demand equation. Shifts in the demand equation are caused by households being able to buy more or better quality food or health services with a given amount of income. Child survival is defined as mortality over the first 10 years of life. The authors use a Weibull survival model estimated with the maximum likelihood estimator.

This paper estimates its model using data from the 1992/1993 and 1997/1998 VLSS. It also divides the births from the sample into two groups - poor (representing the poorest quartile) and rich (representing the other quartiles). This is done through the use of dummy variables. Specifically they include a 1998 dummy variable, a vector showing the interaction between the time dummy variable and the independent variables, a dummy variable indicating whether or not the child is poor and a vector showing the interaction between the poor dummy variable and the independent variables. This paper constrains its estimation so that the coefficients on the independent variables cannot vary over time. The coefficients on the independent variables can be larger or smaller between the two income groups, but not for the same income group between the two periods.

The WB (2002) identifies four groups of variables that could affect child survival. These are regional, child, household and community characteristics. The regional variable accounts for Vietnam's six regions. Child characteristics include gender, child's age and consumption. Household characteristics include a mother's years of schooling, access to adequate water supply, access to sanitation and antenatal visit. Community characteristics include medically trained delivery coverage, facility delivery coverage and vaccination rates.

The main results of the WB (2002) model in terms of which determinants are significant causes of child survival are as follows:

- non-poor children living in the central highlands and southeast have significantly better survival prospects than in other regions;
- poor children living in the central highlands and southeast have worse survival prospects than in other regions;

- gender and age have no significant impact on children's survival whatever the period or income group;
- mother's education improves child survival in both periods and income groups;
- access to adequate water also improves a child's survival prospects;
- sanitation has no significant affect on child mortality among the poor or the non-poor;
- higher vaccination coverage in the child's village also significantly improves chances of child survival;
- antenatal visit coverage significantly reduces child survival among the poor;
- the log of household consumption is also insignificant.

The WB (2002) argues that the above results are broadly in line with expectations and are supported by previous studies. The two exceptions to this are the result that antenatal coverage may reduce child survival and the insignificance of household income. This study argues that household income is insignificant because the effect of consumption appears through the interaction terms. Regarding antenatal coverage it is argued that recent evidence (Furquim De Almeida et al 2000) suggests that home births carry a higher risk but only for poorly educated mothers.

This study concludes by arguing that any strategy for accelerating the decline in child mortality should focus not just on improving coverage of health services and access to water but also on enhancing the impact among the poor of these key determinants of child survival. It also argues that behavioural change programmes could reduce child mortality by enhancing the impact of health services and water supply on child survival among the poor.

WB (2002) is a strong study of the determinants of child mortality which uses nationally representative data from a fairly large sample. Its methodology also allows it to distinguish how the determinants of child mortality affect different income groups. One criticism is that it concludes that behavioural change programmes are important in reducing child mortality even though it does not provide any direct evidence for this.

ADB (2002) examines the determinants of child mortality in Vietnam. This study argues that mother's background, family income, access to health services and gender may all partly determine child mortality in Vietnam. The study examines each of these variables in turn by calculating simple cross tabs, correlations and single variable regression models. Multivariate regression analysis is not used in this paper.

This paper argues that a mother's educational background is strongly related to child mortality. It shows that mothers with no educational experience have almost double the rates of child mortality compared to those who have completed higher secondary education.

A mother's age at the birth of her child is shown to have a U- shaped relationship relative to mortality; with mortality being highest for very young or old mothers. First and higher order births are also shown to be related to higher mortality compared to second or third order births.

ADB (2002) also argues that poverty and household income can be determinants of child mortality. This study shows that province level poverty rates are a statistically significant determinant of infant mortality rates across provinces. The slope of their regression line indicates that a one percentage decrease in the poverty rate will be associated with a decrease of nearly 0.4% in the infant mortality rate.

This paper also argues that access to child health services can affect infant and child mortality. It provides evidence through a single variable regression model that the percentage of measles immunization coverage partly determines infant mortality across regions.

ADB (2002) also briefly discusses the impact of gender on child mortality. This paper argues that while girls have a lower infant and under-five mortality rate than boys in Vietnam this is fully explained by biological advantage. There is, however, some evidence that mortality is higher for girls than boys in the one to four age group and that this may be due to a preference for sons when seeking healthcare. There is also some evidence of a gender bias in immunization coverage; with immunization coverage being 3% to 4% lower for girls than boys, according to data from the 1998 Vietnam DHS.

The ADB (2002) study is a detailed and comprehensive study of the determinants of child mortality. The main problem with this study is that it analyzes each variable in turn and does not examine the impact of each determinant on child mortality when controlling for other variables. This is problematic because many of the determinants it examines may themselves be correlated. Richer families, for example, may also be those where mothers are better educated. It follows that when only mother's education and child mortality are analyzed some of the variation in child survival that is due to income may be falsely proscribed to mother's education.

WHO (2007) discusses child mortality in Vietnam. This study does not undertake a detailed econometric analysis of the causes of mortality and nor does it make a detailed argument linking mortality to specific socioeconomic causes. Rather it presents through cross tabs and graphs simple correlations between child mortality and its possible determinants. These statistics are mainly based on data from the 2002 DHS. The following paragraphs discuss the main conclusions made by WHO (2007).

There are, according to the WHO (2007), no significant differences by gender for neonatal, post-neonatal or infant mortality. Under-three and under-five mortality are higher for males than females and this is probably due to biological advantage. Income appears to be related to child mortality in Vietnam with the richest quintile having approximately half the under-five mortality rate of the poorest quintile. This report also notes that there are significant differences in child mortality by region and ethnic group in Vietnam. Under-nutrition is associated with nearly half of all child deaths according to this report.

WHO (2007) also undertakes a detailed review of the government's health system. This review is not discussed in detail here, as doing so would lead to a great deal of repetition with the later sections of this chapter which discuss social policies in Vietnam. Briefly stated the main conclusions of the WHO (2007) report are that Vietnam has established a dense primary health system, effective programmes to control infectious diseases and effective family planning programmes and that these have contributed to falling and low child mortality in Vietnam.

UNICEF (2010) examines the determinants of child mortality using data from the two VLSS carried out in the 1990s and the 2006 MICS. Using these two data sources allows UNICEF (2010) to identify the determinants of child mortality in the 1990s and mid-2000s and discuss any significant differences in the determinants between these two periods. In all these models child mortality is defined as the proportion of children ever born who have died at any age below 10.

The regression models using VLSS data have two alternative specifications. The first specification includes indicators of mother's nutritional status as an independent variable, while the second does not.

Two alternative statistical models are used. The first is a linear regression model estimated using OLS with the proportion of children ever born who have died as the dependent variable and the cumulative number of each woman's births as a frequency weight. The second is a grouped logit model with the number of child deaths at any age as the number of positive responses and children ever born as the total population. In all, eight models are estimated because there are two VLSS surveys, two alternative specifications and two statistical models.

The explanatory variables used in this model are mother's age, mother's age squared, the highest grade of schooling completed by the mother, the highest grade of schooling completed by any adult household member, a dummy variable indicating membership of the ethnic majority (Hoa or Viet) a living standards measurement and a commune dummy variable to capture fixed effects.

The main results that were consistent across the models estimated were that a mother's age is positively related to mortality and that the highest grade of schooling completed by any household member is negatively related to child mortality. The authors note that it is surprising that the schooling of mothers is not consistently statistically significant, while the highest grade of schooling completed by any household member is significant in all of the eight models estimated. The authors argue that this suggests that in Vietnam any analysis which considers only the schooling of mothers may provide misleading results. A further surprising result is that living standards measurements (either a wealth index or predicted per capita consumption) are not statistically significant in any of the eight models estimated.

UNICEF (2010) also runs regressions using child mortality data from the 2006 MICS. The dependent variable in this model is the proportion of children who have ever died. The estimation sample is women aged 15 to 49 with at least one birth. The explanatory variables are mother's age, mother's age squared, the highest grade of schooling completed by the mother, the highest grade of schooling completed by any adult household member, a dummy variable indicating Chinese or Vietnamese ethnicity, a living standards measurement (either a wealth index or predicted per capita consumption) and a commune dummy variable to capture fixed effects.

The results from this model indicate that the proportion of children who have died at any age is significantly related to mother's age (positively), highest grade of schooling completed by the mother (negatively), highest grade of schooling completed by any household member aged 15+ (negatively) and to ethnicity. The living standards measurements were again found to be insignificant.

The paper argues that these results are as expected and are broadly similar to those obtained using VLSS data. The main difference being that a mother's own schooling is significant in the models that use MICS data. UNICEF et al (2010) argue that this may be because the health education programmes that were targeted at mothers in the late 1990s and early 2000s were more effective among better educated women. The living standards measurement variables are again insignificant, which while surprising is consistent with the earlier results which used data from the VLSS.

This section has reviewed a number of studies of the determinants of child mortality in Vietnam. Our earlier analytical framework argues that family income, mother's education, health outputs and water supply and sanitation will be significant determinants of child mortality. So do the studies reviewed in this section reach the same conclusion?

The studies reviewed in this section do not as a whole provide clear cut evidence that family income is a significant determinant of child mortality. ADB (2002) provides evidence that poverty rates across province and child mortality are associated, but other variables are not controlled for and this study does not establish a relationship between poverty and child survival at the household level. Hoa et al (1997), WB (2002) and UNICEF et al (2010) all find that income is not a significant determinant of mortality. However, Hoa et al (1997) find that morbidity is lower in rich families and the WB study argues that family income affects child mortality through other variables that were found to be significant in its regression analysis. Swenson et al (1995) is the only study reviewed that finds that family income is a significant determinant of child mortality across families when other determinants are controlled for.

There is strong evidence that the education of mothers is a significant determinant of child mortality in Vietnam. ADB (2002) demonstrates that in Vietnam mothers with no educational experience have almost double the rates of child mortality compared to mothers who have completed higher secondary education. Hoa et al (1997) and WB (2002) both find that the education of mothers is a significant determinant of child mortality in Vietnam when other variables are controlled for. These studies do not attempt to differentiate between the power of mothers determining child mortality through maternal factors and control of household resources, and female education affecting mortality through knowledge of specific health interventions.

The studies reviewed also provide evidence that health outputs may partly determine child mortality in Vietnam. Hoa et al (1997) argue that the low levels of child mortality in Vietnam despite high levels of child morbidity may be due to effective local health interventions. The WB (2002) uses higher vaccination coverage in the child's village as a proxy for community level health services and finds that this variable is significant. ADB (2002) provides evidence that child immunizations significantly reduce mortality across provinces. WHO (2007) argues that government health services and programmes have contributed to reductions in child mortality in Vietnam.

The WB (2002) study also finds that access to adequate water is a significant determinant of child mortality in Vietnam. Overall the studies reviewed here support our earlier analytical framework. Specifically they provide evidence that mother's education (although this could be acting as a proxy for power) community level health outputs and water supply and sanitation are significant determinants of child mortality. All these variables were identified as important determinants of child mortality in our analytical framework.

The studies reviewed here also illustrate possible mechanisms through which social policies may be affecting mortality. Government social policies could have reduced child mortality by improving access to clean water, female education and vaccination coverage. If the government's policies in these areas have been more effective than those of the other countries in our sample then this could explain why Vietnam has low child mortality for its structural factors.

5.3 Trends in Child Mortality over Time, its Distribution by Income, Region and Ethnicity and its Epidemiology

Introduction

This section reviews trends in child mortality over time. It also examines the distribution of child mortality by income, region and ethnicity and its epidemiology.

This chapter has three main goals. The first is to gain a better understanding of the trends in, and distribution of, child mortality in Vietnam. The second is to examine whether there is additional evidence to support our view that social policies may explain why Vietnam has low child mortality for its structural factors. The third is to strengthen the causal chain between social policies and child mortality.

Trends in Child Mortality over Time

This section examines child mortality rates over time in Vietnam. In examining trends in child mortality over time the amount of data available is inversely related to the potential accuracy and comparability of the data. If, for instance, we were only to use direct estimates of child mortality data from VLSS for the period immediately preceding the survey then we would have accurate data but we would only have two data points - one from each survey. Alternatively if we used every data point on child mortality in Vietnam available from international databases then we would have infant and child mortality data for many years but these data may be inaccurate and its original source is often unclear. We minimize this problem by initially examining trends in child mortality using household survey data and augmenting this analysis with data from WDI.

Immediate Direct Point Estimates of Infant and Under-Five Mortality Rates

This section analyzes trends in infant and under-five mortality using data from direct point estimates from household surveys. Direct estimates⁴⁶ are calculated by dividing the number of recorded deaths by the number of live births and do not rely on model life tables. Only one data point from each survey is calculated based on the five years immediately preceding the survey. This method provides accurate and comparable data, albeit one with limited data points.

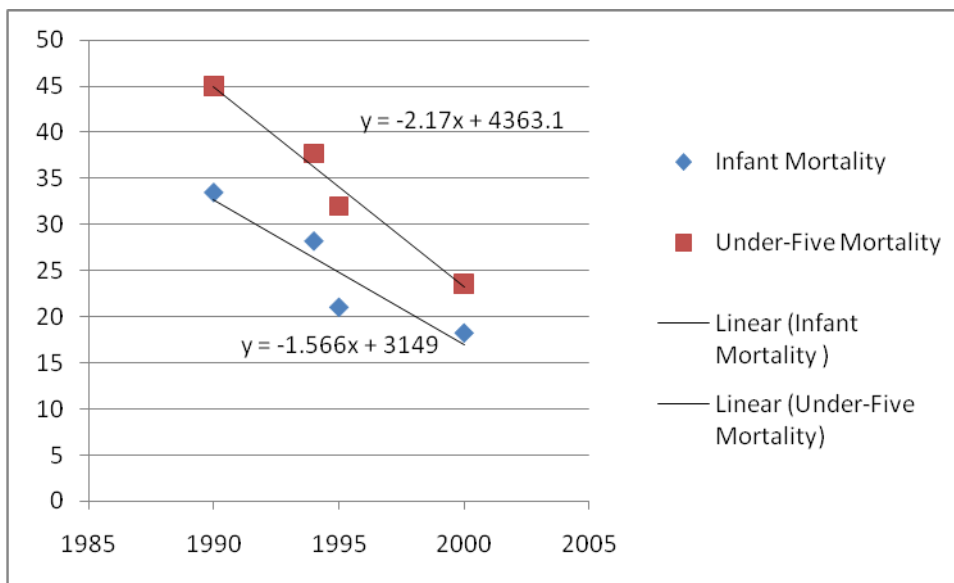
⁴⁶In reality the underlying mathematics are slightly more complex as account has to be taken of children born less than one year preceding the survey (for infant mortality).

According to VLSS data the infant mortality rate declined from 33.5 in 1990 (1992/1993 survey) to 21 in 1995 (1997/1998 survey). This represents a sharp decline of 37% over the first five years of the 1990s. According to DHS data the infant mortality rate declined from 28.2 (1997 survey) to 18.2 (2002 survey) between 1994 and 2000. This represents a sharp decline of 35% over five years. Combining the estimates from the DHS and VLSS surveys shows that infant mortality declined from 33.5 to 18.2 from 1990 to 2000; a decline of 46%.

According to VLSS data under-five mortality declined from 45 to 32 between 1990 (1992/1993 survey) and 1995 (1997/1998 survey). This is equivalent to a 29% decline. According to DHS data the under-five mortality rate declined from 37.7 in 1994 (1997 survey) to 23.6 in 2000 (2002 survey); equal to a 37% decline. Combining estimates from the two surveys shows under-five mortality declined by 48% between 1990 and 2000. This is a sharp decline in under-five mortality.

Overall the data from VLSS and DHS show that under-five and infant mortality sharply declined over the 1990s in Vietnam.

Graph 1: Direct Point Estimates from Five Years Preceding the Survey from DHS and VLSS



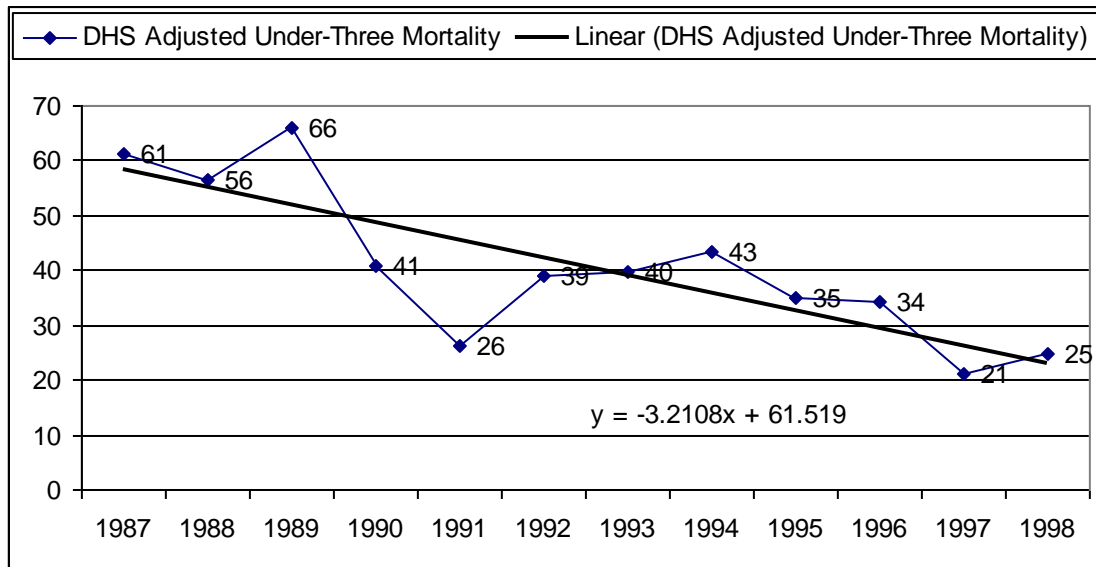
Long term Direct Point Estimates of Child Mortality

The DHS reports provide estimates of infant and under-five mortality for three five year periods before the survey. Using these estimates would increase the number of years for which we have data. The rates of infant and under-five mortality data for more than five years preceding the DHS surveys may, however, be biased downwards because the average age of children's mothers, a significant determinant of child survival, will decline as mortality is calculated for earlier periods (Sahn and Stifel 2003). We avoid this bias by creating our own dataset by recalculating infant and under-three mortality rates from individual mothers' responses to DHS in Vietnam. This gives us seven years of under-three mortality data and nine years of infant mortality data for each DHS survey. When data points overlap we take the simple average of the values from the two surveys. We did not calculate under-five mortality rates as this would lead to just five years of data per survey. However, as there is a

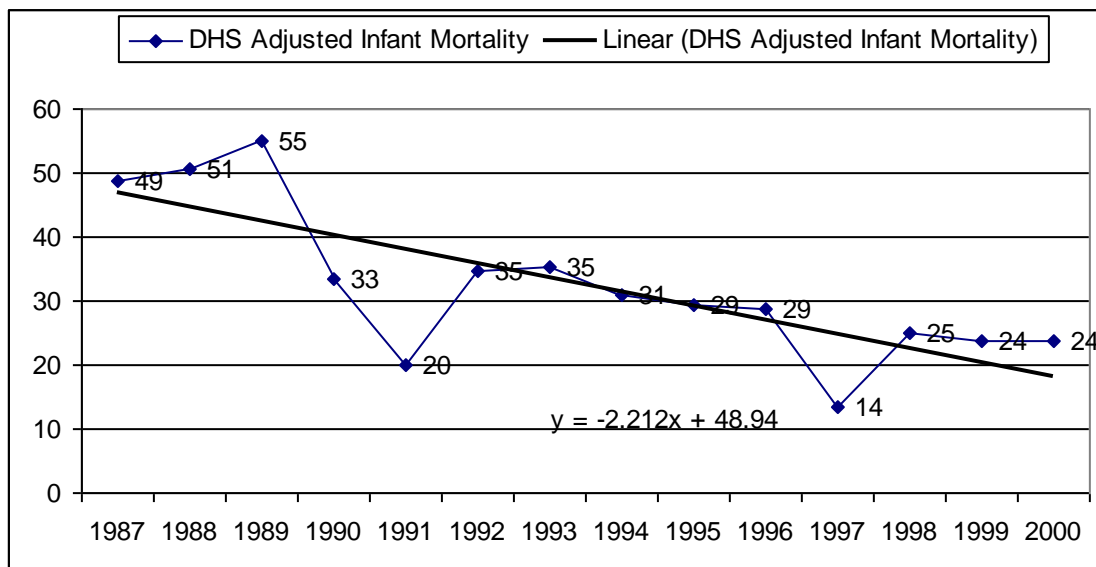
strong correlation between under-three and under-five mortality this is arguably not a serious omission.

Graph 2 shows that under-three mortality in Vietnam increased in the late 1980s before sharply declining from 1990 until 1998. The under-three mortality rate fell from 66 in 1989 to 25 in 1998; a decline of 62%. Infant mortality also increased in the late 1980s before sharply declining throughout the 1990s. From 1989 to 2000 the infant mortality rate declined from 55 to 24; equivalent to a 56% decline. This is a sharp decline in infant mortality.

Graph 2: DHS Adjusted Under-three Mortality Data



Graph 3: DHS Adjusted Infant Mortality Rate

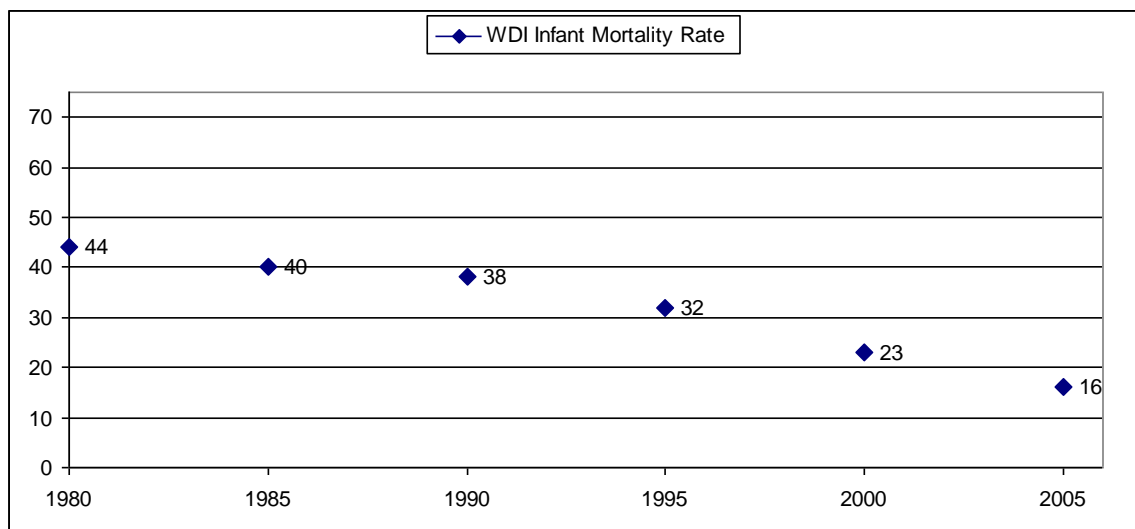


International Dataset Estimates of Infant and Child Mortality

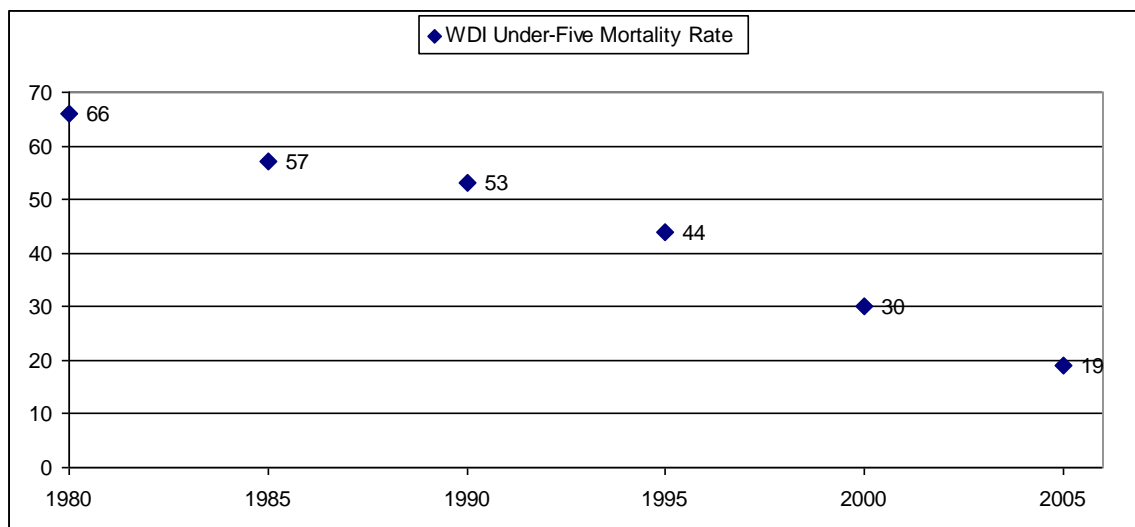
Our earlier regression analysis in which Vietnam was an outlier included infant and under-five mortality data from WDI until 2006. The next two graphs show the infant and under-five mortality rates using WDI. These data also show a sharp decline in infant and under-five mortality throughout the 1990s. This decline continued into the 2000s with infant mortality declining from 23 to 16 and under-five mortality declining from 30 to 19 from 2000 to 2005. This is a substantial decline in under-five mortality.

This rate of decline into the 2000s can also be confirmed using estimates of infant mortality from the 2006 MICS. This data source shows that the infant mortality rate had fallen to 22 by 2003. This compares to a rate of 24 in 2000 from our DHS Adjusted data.

Graph 4: WDI Infant Mortality Rate in Vietnam



Graph 5: WDI Under-five Mortality Rate in Vietnam



Overall data from a variety of sources show that infant, under-three and under-five mortality in Vietnam sharply declined during the 1990s and early 2000s.

Trends in Child Mortality in Vietnam in Comparative Perspective

The statistical analysis conducted in appendix 13 shows that the rates of decline in infant and under-three mortality in Vietnam during the 1990s were much higher than those seen in most other countries. Vietnam is also one of the few developing or middle-income countries that will, based on current trends, achieve the fourth MDG.

This conclusion was supported by other studies that used different statistical techniques and data. The Child Survival (2005) report concluded that Vietnam was one of only 7 of the 60 developing countries examined that were on track to achieve MDG4. Murray et al (2007) concluded that Vietnam was one of only 32 of 172 countries examined that had a greater than 75% chance of meeting MDG4.

Conclusion

Vietnam has seen sharply declining rates of infant, under-three and under-five mortality. These are of course not necessarily due to effective social policies. It could be that structural factors such as economic growth have driven reductions in child mortality. Yet it would be counterintuitive if the sharp reductions in child mortality experienced by Vietnam occurred while its social policies deteriorated. In addition, as we have already seen, structural factors only partly explain why Vietnam has such low child mortality compared to other countries.

The fact that Vietnam is, based on current trends, likely to achieve MDG4 also makes it an interesting case study. For if declines in mortality in Vietnam were associated with effective social policy then this indicates that the governments of the developing world can reduce child mortality through public policy. Yet if Vietnam is only achieving MDG4 due to changes in its structural factors beyond the immediate control of its government, then this calls into question the use of developing countries committing to international goals. There is no logic to holding governments to account for targets of which the determinants are beyond their control.

Distribution of Child Mortality by Household Expenditure

Introduction

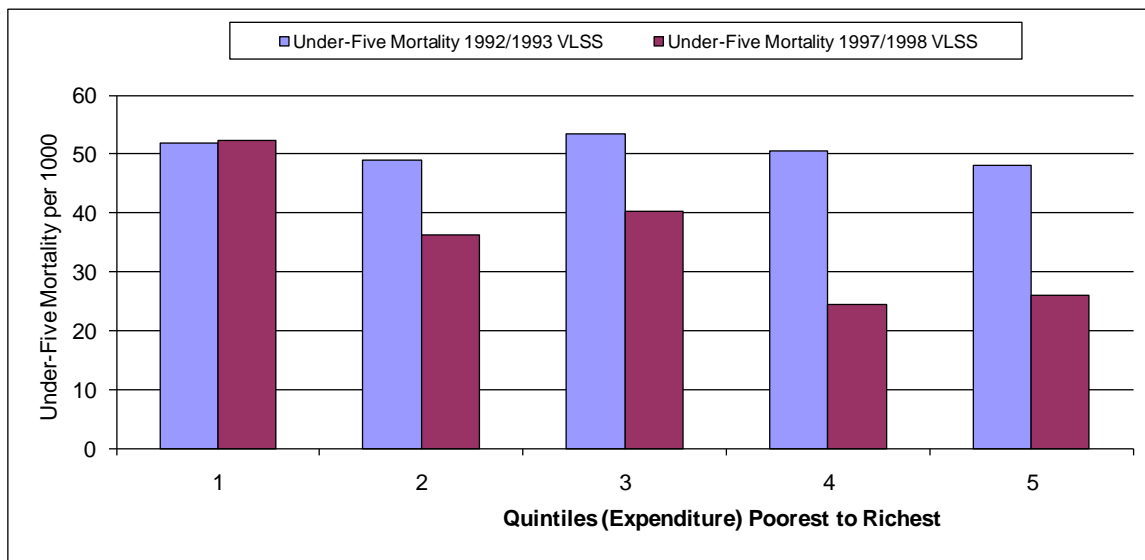
This section discusses the distribution of child mortality by household expenditure in Vietnam. It analyzes trends in inequality in child mortality by expenditure over time, possible causes of these trends and compares the distribution of child mortality by expenditure in Vietnam to that of other countries. We also discuss whether the distribution of child mortality by expenditure in Vietnam compared to other countries can explain why it has low child mortality for its structural factors.

This section uses data drawn from the 1992/1993 and 1997/1998 VLSS. These datasets are used because direct estimates of child mortality and family expenditure can be calculated from them. Data from the two DHS are also used but to a lesser extent as these surveys only include data on family assets and not expenditure. Some estimates are also taken from the 2006 MICS.

Trends in the Distribution of Child Mortality by Expenditure over Time

Population weighted quintiles are a widely used measure to demonstrate inequalities in health outcomes. Graph 6 shows population weighted quintiles for under-five mortality where the unit defining the quintile is live births and the measure of living standards is per capita expenditure. It demonstrates that between the two VLSS there was a substantial increase in inequality in under-five mortality. In the first period households in the richest quintile had an under-five mortality rate which was just 7% lower than the poorest quintile, but by the second period this had grown to being 50% lower. This indicates that the sharp reductions in under-five mortality in Vietnam in the early and mid 1990s were not spread evenly across the population but were concentrated among the better off.

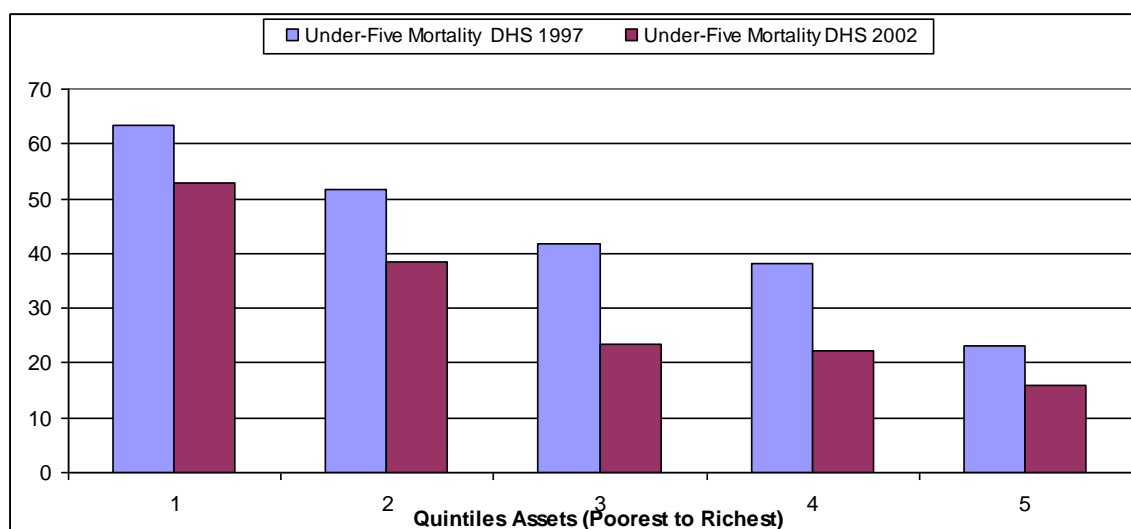
Graph 6: Under-Five Mortality by Expenditure Quintile VLSS



Quintiles for under-five mortality can also be calculated using DHS data. These quintiles are defined by an asset index (because DHS does not include data on income or expenditure) and are thus not directly comparable to those estimates produced using VLSS data. Graph 7 shows the quintile data produced from DHS. These estimates are for the five years preceding the year of the survey.

The DHS data show that the largest decline in mortality in absolute terms was in the third and fourth quintiles. The fifth quintile saw the smallest absolute decline in mortality, but as mortality declines more slowly over time from low rates, and under-five mortality among the richest quintile was an already low 23 in the 1997 DHS, this is not surprising. In percentage terms the first and second quintiles saw the smallest decline followed by the fifth quintile; with the third and fourth quintiles showing the largest declines. The DHS data show increased income inequality over time; with this inequality being driven by the third and fourth quintiles seeing larger declines in mortality than poorer households.

Graph 7: Under-five Mortality by Asset Quintile DHS



The relationship between child mortality and family assets can also be studied using a concentration curve equation. This equation calculates the cumulative distribution of mortality compared to the cumulative number of families ordered from poorest to richest. For under-five mortality zero indicates complete equality; that is whatever a family's income, their children face the same chance of dying. A minus number shows that richer families face less chance of their children dying than poor families and a positive number indicates that poor families face less chance of their children dying. Table 19 shows concentration curve values in Vietnam over time (from different datasets). The concentration curves are calculated using asset indexes (because DHS data do not contain consumption or income data).

Table 19: Concentration Curve Index in Vietnam over Time⁴⁷

Concentration Curve Index Under-Five Mortality	
VLSS (1992/1993) Assets	-0.103
DHS (1997) Assets	-0.159
DHS (2002) Assets	-0.232

The interpretation of table 19 is relatively clear cut. Inequality in under-five mortality by family assets increased in Vietnam throughout the 1990s. By the early 2000s Vietnam had highly stratified under-five mortality by income. Overall the evidence from VLSS and DHS data is that inequality in under-five mortality increased throughout the 1990s.

Studies of the Causes of Inequality in Child Mortality in Vietnam

This section reviews studies from WB (2002) and UNICEF (2010) which seek to explain the increase in inequality in child mortality in Vietnam.

The WB (2002) examines the determinants of increased inequality in child mortality by family expenditure. It defines child mortality as death during the first 10 years of life and

⁴⁷ Concentration curve equations taken from UNICEF (2010) and Gwatkin et al (2007).

uses data from the VLSS of 1992/1993 and 1997/1998. This study divides the sample of families into two groups – poor and non-poor. The poor group refers to the poorest quartile of families by consumption. The non-poor group refers to families in the three richest quartiles by consumption. A demand equation is then specified linking child survival to household resources and other variables affecting the “shadow price” of child survival. A Weibull survival model is then used to estimate the model.

This model is specified so that there can be three reasons for the poor showing smaller declines in their mortality. First, the poor could experience less beneficial changes in the means of the underlying determinants of child mortality. Second, the coefficient mediating the relationship between the determinant and child mortality could be worse for the poor than the non-poor. Third, changes in the coefficients over time may matter less to the poor since they had worse underlying determinants of child mortality in the initial period. A fourth effect, namely differential changes in the coefficients of the poor and non-poor, is constrained in the model.

Four groups of variables that could affect child survival are identified in this study. These are regional, child, household and community characteristics. Child characteristics include gender, child's age and household consumption. Household characteristics include year of schooling, access to adequate water supply, access to sanitation and antenatal visit. Community characteristics include medically trained delivery coverage, facility delivery coverage and vaccination rates.

This paper's results show that the poor experiencing less advantageous changes in the determinants of child mortality and the poor facing worse coefficients on these determinants played a roughly equal role in explaining why the survival prospects of poor children did not increase by as much as the non-poor.

Regarding changes in the determinants of child survival, vaccination coverage, antenatal visits and facility deliveries, all increased more among the non-poor than the poor. Access to clean water also increased faster among the non-poor than poor. Mother's education, meanwhile, actually declined among the poor while increasing among the better off.

Regarding the poor facing worse coefficients, immunization, antenatal coverage and clean water have a larger impact among the non-poor. So a unit increase in any of these variables results in a larger increase in child survival among the non-poor than the poor.

This paper also finds that changes in the coefficients over time partly explain increased inequality between the poor and the non-poor. However, with this type of variation no single variable is statistically significant in explaining increased inequality.

One criticism of the WB study is that it does not identify why immunization, antenatal coverage and clean water have a larger impact on the child mortality of the non-poor. These determinants must be interacting with some intermediate variable such as a mother's knowledge of health interventions. This suggests that the intervening variables should be identified and included in the econometric model. Simply stated if vaccination coverage has a smaller impact on the poor than the non-poor then we need to identify why this is the case.

UNICEF (2010) investigates inequality in child mortality. This study defines mortality among children born from the 1992/1993 VLSS as the health outcome of interest. The study

seeks to explain the underlying causes of inequality in this variable as defined by the concentration curve equation with household consumption as the living standards measurement. It does this by first undertaking a regression analysis to find the variables that are most closely associated with mortality among children born. The results of this regression and its methodology were discussed earlier in this chapter and are not repeated here.

This study then uses the results from its regression analysis to decompose the concentration curve equation. This analysis takes account of the strength of the variable's association with child mortality and the variable's own distribution across income groups. This study's results show that a mother's schooling, highest secondary level schooling of a parent, ethnicity and the impact of wealth on mortality explain inequality in child mortality.

The analysis undertaken by UNICEF can be criticized in a number of regards. First, the study explains inequality in mortality using the VLSS of 1992/1993. This is problematic as there was not a great deal of inequality in child mortality in that survey. Second, as only one survey is analyzed the results do not explain why inequality in child mortality increased in Vietnam during the 1990s. Third, the methodology used means variables that were found to be insignificant as causes of mortality can, because of their distribution across income, be found to be significant causes of the inequality in mortality. This leads to some counterintuitive results such as the paper arguing that wealth does not significantly determine child mortality but does significantly determine inequality in child mortality.

In conclusion the WB (2002) and UNICEF (2010) studies provide highly complicated, but in our opinion not entirely convincing, answers to the question of what explains inequality in child mortality in Vietnam. The WB (2002) study is problematic because it shows that some determinants of child mortality have a bigger impact among the non-poor than poor but does not explain why this is the case. The UNICEF (2010) study is problematic because it does not explain why inequality in mortality increased in Vietnam in the 1990s and states some counterintuitive results.

Statistical Analysis of the Causes of Increased Inequality in Child Mortality

This section undertakes a brief statistical analysis of the causes of increased inequality in child mortality. Our earlier analytical framework identified the following main causes of child mortality: family income, mother's education and power, health outputs and water supply and sanitation. Increased inequality in these determinants of child mortality in the 1990s could explain the increased inequality that we have observed.

The next table shows concentration curve equations for these different determinants of child mortality from different household surveys. The 1992/1993 VLSS already showed significant inequality in the determinants of child mortality. Child malnutrition and total fertility were higher among the poor in this survey. In contrast, family planning utilization, antenatal care and child immunization were all more common among richer families.

Table 20: Inequality in the Determinants of Child Mortality

	VLSS 1992/1993	1997 DHS	2002 DHS	2006 VHLS	2006 MICS
Source	UNICEF (2010)	Gwatkin et al (2007)	Gwatkin et al (2007)	UNICEF (2010)	UNICEF (2010)
Child Malnutrition / (underweight or stunting)	-0.093	Not Calculated	Not Calculated	-0.198	Not Calculated
Total Fertility Rate	-0.079	-0.141	-0.090	Not Calculated	-0.074
Family Planning Utilization Modern Method	0.102	0.014	-0.025	Not Calculated	-0.008
Antenatal Care	0.125	0.115	0.009	Not Calculated	0.059
Child Immunization Full Basic Coverage	0.147	0.065	0.117	Not Calculated	0.108
School Completion Among Women	Not Calculated	0.123	0.107	Not Calculated	Not Calculated

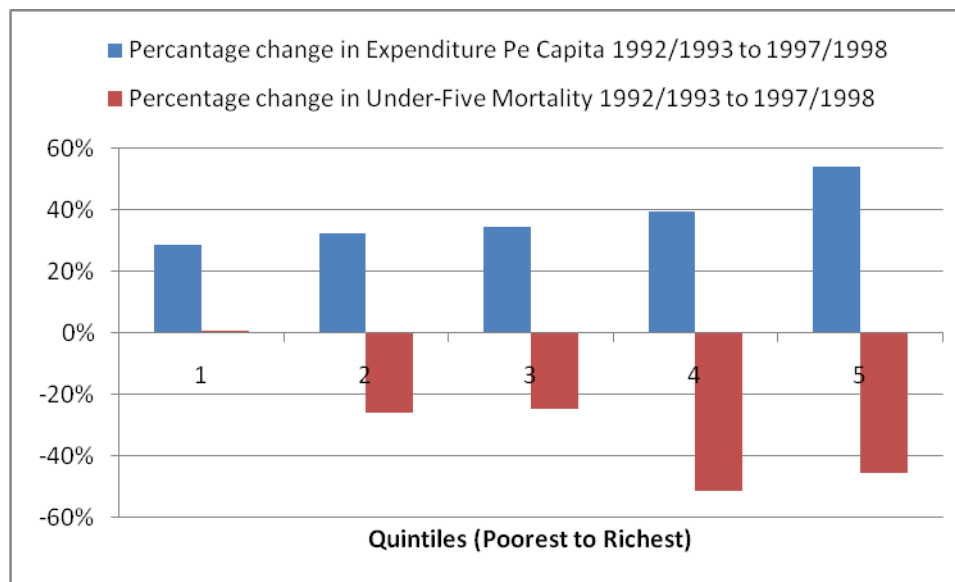
The trend in the degree of inequality among the determinants of child mortality over time is more difficult to discern. The distribution of child malnutrition became more unequal between the 1992/1993 VLSS and the 2006 VLSS. The total fertility rate may have become more unequal in the early and mid-1990s before becoming slightly less unequal in the early 2000s. Family planning utilization became consistently less unequally distributed through the 1992/1993 VLSS, 1997 DHS and 2002 DHS. By the 2002 DHS family planning utilization was favouring poorer families. Antenatal coverage became more equally distributed over time until slightly increasing in the 2006 MICS. Child immunization coverage inequality decreased between the VLSS 1992/1993 and the 1997 DHS before increasing in the 2002 DHS and then slightly decreasing in the 2006 MICS. Inequality in school completion among women slightly decreased between 1997 and 2002.

These trends in the distribution of the determinants of child mortality should be interpreted cautiously. The differences in the concentration curve equations are small and the trends are often not consistent from 1992/1993 to 2006. In addition the declines in inequality between the 1992/1993 VLSS and the 1997 DHS for family planning, antenatal care and child immunization occurred while inequality in child mortality increased. Indeed only total fertility inequality increased from 1992/1993 to 1997 despite inequality in mortality by income increasing during the early and mid-1990s. It thus seems unlikely that increased inequality in the non-income determinants of child mortality shown in table 20 can explain the increase in inequality in child mortality in the 1990s.

Increased income inequality could itself account for the increase in inequality in child mortality seen between different income quintiles. If richer quintiles saw a much larger increase in their income than poor quintiles then this may itself explain why the rich saw a larger decrease in child mortality than poorer families.

The next graph shows the percentage change in expenditure per capita and child mortality using data from the 1992/1993 and 1997/1998 VLSS by quintile. The data show that richer quintiles saw a much larger increase in income and a much larger decline in child mortality than the poorer quintiles. This evidence suggests that the increase in inequality in child mortality by quintile seen in Vietnam occurred because richer quintiles saw a larger increase in income than poorer quintiles.

Graph 8: Percentage Change in Income and Mortality by Quintile (VLSS Data)



The liberalization of the health and education sectors, discussed later in this chapter, could also have contributed to greater inequality in child mortality by strengthening the link between income and child mortality. However, if this was the case, we would expect some outputs from the health system such as child immunization to show greater inequality, but as discussed earlier this was not the case.

The Distribution of Child Mortality and its Determinants by Income in Vietnam in a Comparative Perspective

This section compares inequality in under-five mortality in Vietnam and other countries. We would expect inequalities in under-five mortality to be greater in countries where social policy favoured the rich than in countries with egalitarian or redistributive health and education policies. It may also be the case that egalitarian social policy leads to a country having a low overall rate of child mortality for its structural factors because there are diminishing marginal returns between health and education outputs and child mortality. An equal distribution of child mortality between rich and poor families and egalitarian social policies could then explain why Vietnam has low child mortality for its structural factors.

Table 21 compares inequality in under-five mortality in Vietnam to other countries. All countries shown had a GDP per capita of between 75% and 125% of Vietnam's in 2005. The concentration curve equations in the next table are taken from Gwatkin et al (2007) and are based on DHS data.

Table 21: Concentration Curve Equations for Vietnam and Countries with a Similar GDP

	Year of DHS Survey	Concentration Curve Equation	SE
Uzbekistan	1996	-0.057	0.00192
Pakistan	1991	-0.084	0.00109
Cote d'Ivoire	1994	-0.115	0.0014
Kyrgyzstan	1997	-0.115	0.0018
Cameroon	2004	-0.130	0.04063
Yemen	1997	-0.138	0.00059
Vietnam	1997	-0.159	0.002
Indonesia	2002/2003	-0.189	0.07137
Nicaragua	2001	-0.196	0.00118
Vietnam	2002	-0.232	0.002
Mauritania	2000/2001	-0.278	0.00115

The data show that the inequality in under-five mortality according to income was higher in Vietnam than in most other comparable countries. Vietnam had the fourth highest level of inequality if the DHS from 1997 is used and the second highest if data from the 2002 survey are used. This suggests that Vietnam's low child mortality for its structural factors cannot be explained by egalitarian social policy; because in a country with equal access to social policy outputs we would expect there to be less and not more variation in child mortality by income group.

We can also examine concentration curves of the outputs from social policy. The next table shows concentration curves for antenatal care, child immunization and school completion for women. These social policy outputs are likely to be significant determinants of child mortality. Overall, according to these measures, Vietnam appears to have significantly less egalitarian social policy than Uzbekistan and Kyrgyzstan; approximately as egalitarian social policy as Indonesia, Nicaragua and Cameroon and more egalitarian social policy than Cote d'Ivoire, Mauritania, Yemen and Pakistan. There is therefore little evidence that Vietnam has significantly more egalitarian social policies than other countries.

Table 22: Concentration Curve Equations for Vietnam and Comparable Countries

	Year of DHS Survey	Child Immunization Full Basic Coverage	Antenatal Care	School Completion Among Women	Simple Average
Uzbekistan	1996	-0.012	0.008	0.001	-0.001
Kyrgyzstan	1997	0.001	0.003	0.002	0.002
Indonesia	2002/2003	0.127	0.050	0.079	0.086
Vietnam	2002	0.118	0.073	0.107	0.099
Vietnam	1997	0.065	0.115	0.123	0.101
Nicaragua	2001	0.044	0.067	0.240	0.117
Cameroon	2004	0.103	0.081	0.209	0.131
Cote d'Ivoire	1994	0.126	0.092	0.348	0.189
Mauritania	2000/2001	0.170	0.190	0.403	0.254
Yemen	1997	0.362	0.288	0.514	0.388
Pakistan	1991	0.206	0.482	0.559	0.416

The analysis of health and education policies conducted later in this chapter confirms that access to these services is partly determined by family income in Vietnam, and that the extent to which family income determined the quality of care received, increased throughout the 1990s. Vietnam's low child mortality for its structural factors may be explained by effective social policy but it is not explained by egalitarian social policy.

Conclusion

Inequality in child mortality by family expenditure increased in Vietnam in the 1990s. Child mortality is now as stratified by income in Vietnam as in many other developing countries. The reasons for this increased stratification by income are difficult to discern. We do not find previous studies of this issue to be convincing and our own analysis shows that many of the determinants of child mortality did not become more stratified by income. The increase in inequality in child mortality between income quintiles may be occurring simply because richer quintiles saw a larger increase in their income than did poorer quintiles. Social policy outputs are not more equally distributed in Vietnam than in comparable countries and egalitarian social policies cannot explain why it has low child mortality for its structural factors.

Regional Distribution of Child Mortality

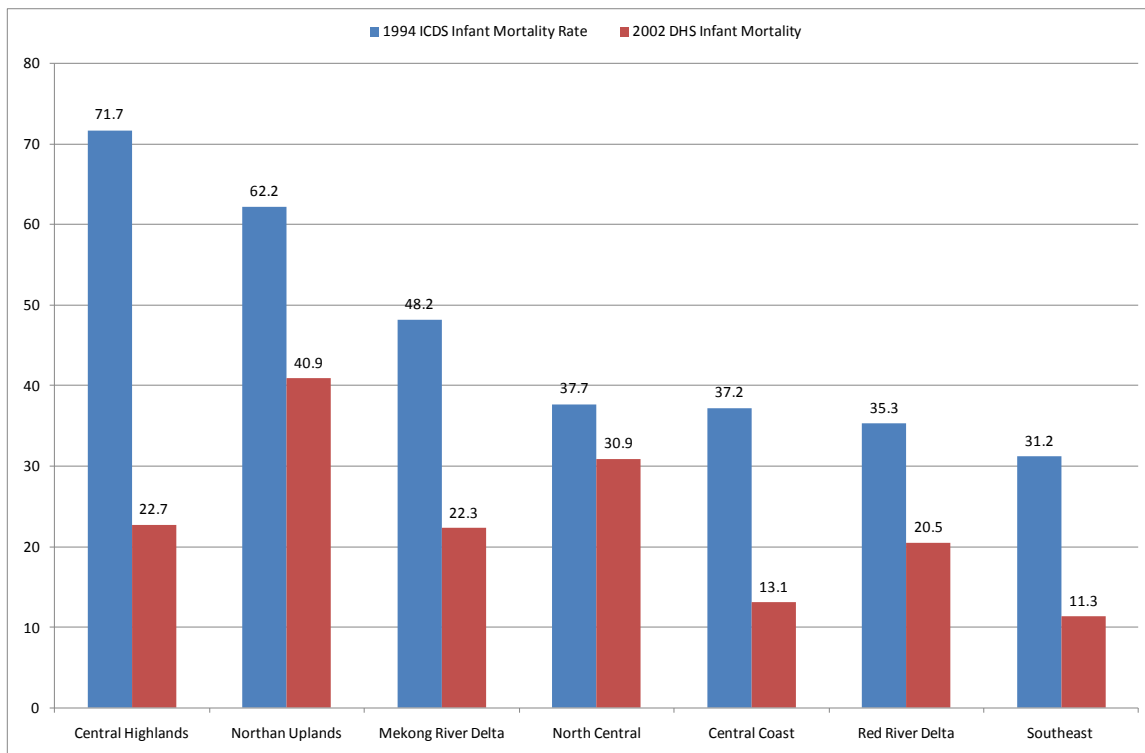
This section discusses the distribution of child mortality by region in Vietnam. It presents estimates of child mortality by region and undertakes a brief statistical analysis of the determinants of regional child mortality.

Child Mortality Rate by Region

Vietnam can be divided into seven regions. Graph 9 shows the infant mortality rate by region based on the 1994 ICDS and the 2002 DHS. These two surveys were used because

they provide accurate estimates of infant mortality over a period that is highly relevant to our analysis. Infant, and not under-five, mortality was used because it is measured over one year and therefore gives an indication of how mortality changed from the mid-1990s to the early 2000s. This fits better with the period of our analysis than under-five mortality rates would.

Graph 9: Regional Infant Mortality in Vietnam

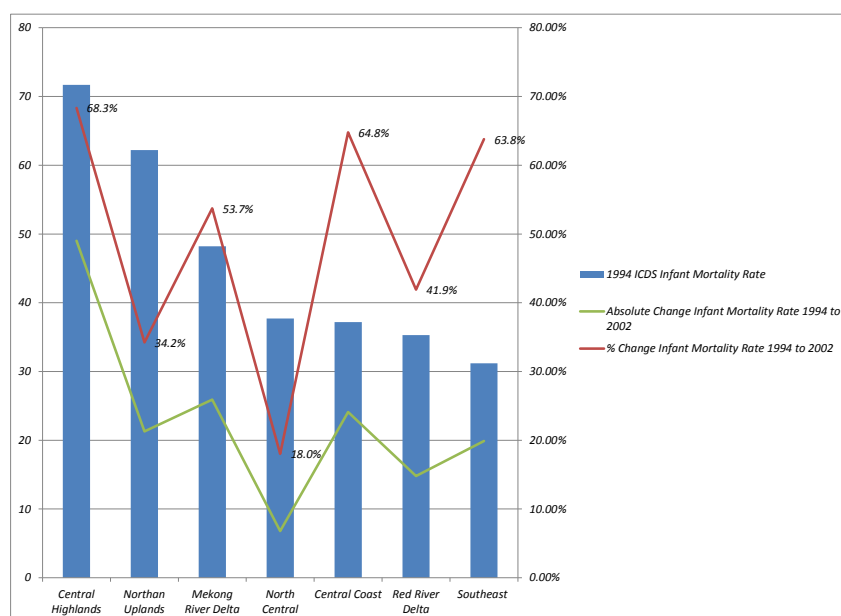


We can delineate three key points from graph 9. First, the distribution of infant mortality between regions was highly unequal in the 1994 ICDS. Infant mortality in the Central Highlands was more than double that of the South East region. Second, infant mortality declined in every region, but some regions saw much larger declines than others. The Central Coast saw its mortality decline by 67% compared to a decline of just 18% in the North Central Region. Third, variation in infant mortality by region has increased over time. The coefficient of variation for regional mortality increased from 0.33 to 0.44 from the 1994 ICDS survey to the 2002 DHS; indicating that the distribution of mortality by region has become more unequal over time.

As discussed earlier, declines in infant mortality may be non-linear over time. It may be easier to reduce infant mortality from 60 to 40 than it is to reduce it from 30 to 10. There may also be diminishing marginal returns between infant mortality and many of its determinants. An increase in family income from \$600 a year to \$800 may reduce infant mortality by more than an increase from \$20,000 to \$20,200 dollars. This suggests that regions with an initially high rate of infant mortality may see larger declines in infant mortality. This has, however, not been the case in Vietnam (graph 10). The Central Highlands initially had a high rate of infant mortality and saw a large decline. However, the North Central region had a high rate of

infant mortality in 1994 but saw a smaller decline than the Central Coast and South East regions.

Graph 10: Change in Regional Mortality Rates in Vietnam



The next table compares infant and under-five mortality rates by region. As expected there is a strong correlation between these two measures and significant variation in the under-five mortality rate by region.

Table 23: Infant and Under-five Mortality by Region

Region	2002 DHS Infant Mortality	2002 DHS Under-Five Mortality
Central Coast	13.1	15.9
Southeast	11.3	22.8
Red River Delta	20.5	26.3
Mekong River Delta	22.3	30.9
North Central	30.9	36.3
Central Highlands	22.7	40.9
Northern Uplands	40.9	51.8
Coefficient of Variation	0.44	0.37
Correlation Coefficient	0.91	

Regional variation in under-five mortality in Vietnam can also be compared to that of other countries. The next table makes this comparison by showing the coefficient of variation in regional under-five mortality for Vietnam and other countries with a similar GDP per capita. According to this measure regional inequality in under-five mortality is higher in Vietnam than in all other countries in the sample bar Cameroon.

Table 24: Coefficient of Variation for Under-five Mortality by Region in Vietnam and Countries with a Similar GDP per Capita⁴⁸

	Coefficient of variation for regional under-five mortality DHS
Cameroon	0.39
Vietnam	0.37
India	0.36
Nicaragua	0.30
Mauritania	0.25
Sudan	0.21
Cote d'Ivoire	0.20
Pakistan	0.15

Statistical Analysis of the Determinants of Child Mortality by Region

This section seeks to explain the regional variation in child mortality. The methodology employed in this section is to examine the relationship between regional child mortality and each of its likely determinants using single variable OLS regression analysis. Multivariate regression analysis is not used because the limited number of regions makes such an analysis problematic.

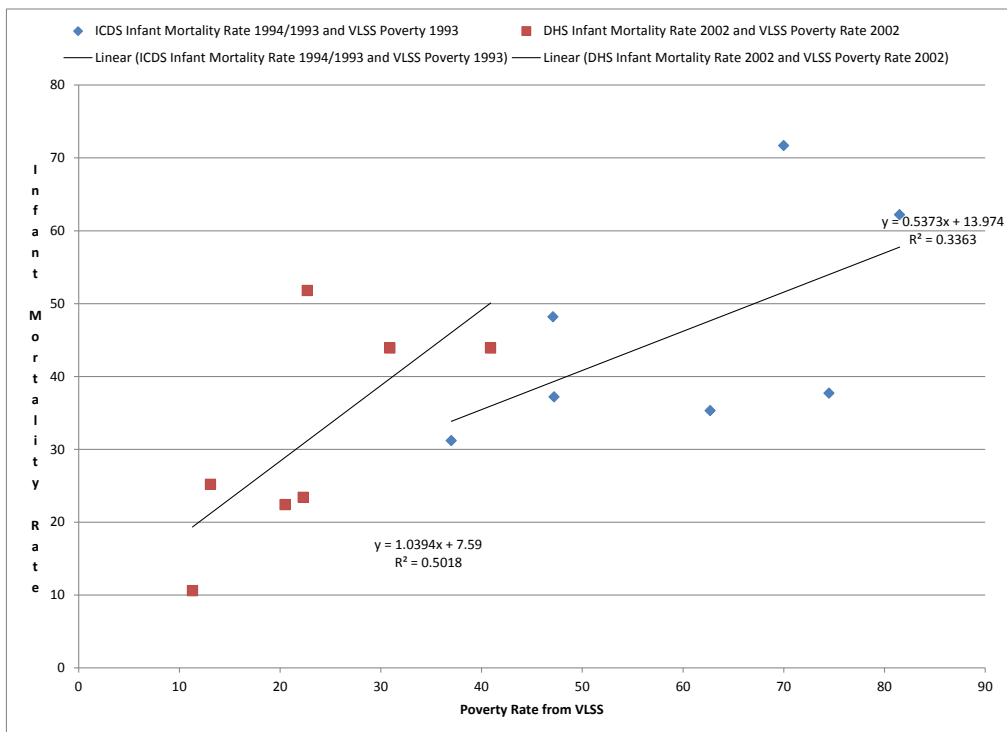
Our earlier analytical framework identified the determinants of child mortality. Based on this framework possible determinants of regional child mortality may include: family income, female education, health outcomes and water supply and sanitation.

This section uses poverty as opposed to per capita income to explain regional differences in infant mortality. The logic behind this decision is that there may be diminishing marginal returns between income and infant mortality. This suggests that increases in income for those on low incomes – which would be reflected in a decrease in the poverty rate - may be more powerful in explaining infant mortality than average per capita expenditure, which could be driven by increases in income among the rich.

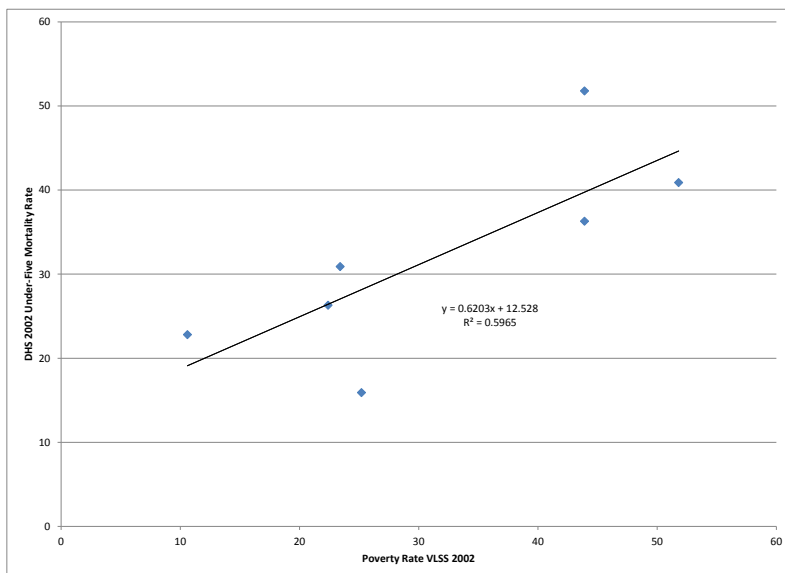
Graph 11 shows, using VLSS poverty data, that in 2002 there was a strong relationship between poverty and infant mortality. Regions with less poor people tended to have lower rates of infant mortality. The association between poverty and infant mortality was weaker in 1993/1994. This may be because as the health system was liberalized in Vietnam the ability of families to pay became a stronger determinant of access to the healthcare. It may also be because fiscal decentralization in Vietnam in the 1990s led to higher per capita spending on healthcare in richer regions compared poorer ones (WHO 2003). As shown in graph 12 there is also a strong association between poverty and under-five mortality by region.

⁴⁸ Author's own calculations of the coefficient of variation based on regional mortality data from different countries' DHS. The surveys all use data from between 1995 and 2005, apart from Sudan, which dates from 1990.

Graph 11: Infant Mortality Rate and Poverty across Regions



Graph 12: Under-five Mortality Rate and Poverty



The rest of this section uses under-five mortality. The reason for this is that many of the determinants we examine, such as women's education and vaccinations, may have a more significant affect on under-five mortality than on infant mortality.

Table 25 shows under-five mortality by region and its likely determinants. This table shows that poverty across regions is highly correlated with, and is a significant determinant of, under-five mortality by region. In contrast neither the education of mothers nor their knowledge of specific health interventions (measured by knowledge of Oral Rehydration Therapy) are significant determinants of the variation in under-five mortality across regions. Oral Rehydration Therapy was chosen as an independent variable because it is a cost effective way to treat diarrhoea, which as discussed in the epidemiological section of this thesis, is a significant cause of under-five mortality in Vietnam.

Table 25: Under-five Mortality and Possible Determinants by Region

	2002 DHS Under-Five Mortality	VLSS 2002 Monetary Poverty Rate by Region	2002 DHS % of Married Women Completed Primary School or Better	2002 DHS Percentage of Mothers with Birth in Last Three Years with Knowledge of ORT	2002 DHS % of Married Women Unmet Need for Family Planning	2002 DHS Percentage of Children who Received all Relevant Vaccinations	2002 VLSS Access to Improved Water Source
Central Coast	15.9	25.2	77.8	66.9	4.4	76	94.635
Southeast	22.8	10.6	74.7	65.8	5	76	97.46
Red River Delta	26.3	22.4	97.8	82.5	3.1	88.4	71.38
Mekong River Delta	30.9	23.4	51.1	73.4	3.7	60.8	49.17
North Central	36.3	43.9	88.2	74.8	6.2	55.9	92.6
Central Highlands	40.9	51.8	61.4	45.3	12.3	55.9	89.15
Northern Uplands	51.8	43.9	69.8	60.5	6	45.1	73.285
Correlation with under-five mortality		0.77	-0.28	-0.43	0.50	-0.85	-0.25
Intercept	N/A	12.528	47.818	61.16	20.74	76.23	45.98
Coefficient on independent variable	N/A	0.62	-0.2108	-0.4331	1.958	-0.6739506	-0.1709
R-Squared	N/A	0.5965	0.076	0.1853	0.2509	0.7162	0.0614
P-Value	N/A	0.04	0.549	0.335	0.252	0.016	0.592

Family planning can reduce infant mortality by reducing total fertility and increasing birth spacing. Differences in family planning by region in Vietnam may partly explain the variation in under-five mortality by region. A common method for measuring the effectiveness of family planning programmes is to measure the unmet need for family planning. Currently married women who are fecund and who do not want any more children or do not want another child for at least two years but who are not currently using contraception are said to have an unmet need for family planning. Table 25 shows that there is only a weak association between the unmet need for family planning and under-five mortality across regions. This suggests that differences in the efficacy of government family planning programmes by region cannot explain regional variation in mortality in Vietnam.

Childhood vaccinations could also explain the variation in under-five mortality. Table 25 shows that there is a strong association between the percentage of children who have received a complete set of vaccinations and under-five mortality across regions in Vietnam. In an OLS regression model with under-five mortality as the dependent variable and the percentage of children who had received a complete set of vaccinations as the independent variable the R-squared was 0.72 and the coefficient on the independent variable was significant at the 5% level.

Access to an improved water source can also reduce under-five mortality. The results in table 25, however, show that this determinant explains little of the regional variation in under-five mortality.

In summary, according to the statistical analysis undertaken in this section the monetary poverty rate and vaccinations are the most likely determinants of under-five mortality across regions. These results should, however, be interpreted with a degree of caution because the limited number of regions in Vietnam indicate that our statistical analysis is undertaken with just seven observations.

The finding that the percentage of children who received vaccinations is a significant determinant of regional under-five mortality should also be interpreted cautiously. Many child vaccinations are provided through the public health system in Vietnam and it is reasonable to assume that regions with high rates of vaccinations may also have better health outputs in other areas. DHS data show, for example, that vaccination rates are strongly and significantly correlated with the number of births attended by a trained health professional. This means that vaccinations may be best interpreted as a proxy for a range of health outputs that reduce under-five mortality.

Conclusion

There are substantial variations in infant and under-five mortality across regions in Vietnam. Inequality in regional mortality has increased over time. Poverty is one significant determinant of this variation in regional mortality. The relationship between poverty and infant mortality has strengthened over time possibly due to family income increasingly determining access to health services. This point is discussed in greater detail later in this thesis. Vaccinations and other health outputs may also explain some of the variation in under-five mortality across regions.

Distribution of Child Mortality by Ethnicity

Introduction

Vietnam is an ethnically diverse nation. Schliesinger (1997) argues that it is one of the most ethno-linguistically and ethno-culturally complex countries in the world. Different ethnic groups have migrated to Vietnam at different times, some many thousands of years ago and others in the last few centuries (Schliesinger 1998); resulting in a heterogeneous cultural and linguistic mix.

The official government classification recognizes 54 different ethnic groups. The Kinh (also called Viet) is by far the largest ethnic group consisting of 86.2% of the population according to the 1999 census of Vietnam. This ethnic group dominates the culture and economy of Vietnam. Table 26 shows the population of the 10 largest groups in Vietnam according to the 1999 census.

Table 26: Population of Vietnam by Ethnic Group

Ethnic Group	Population	% of Total Population
Kinh	65.80	85.8%
Khmer	1.06	1.4%
Muong	1.14	1.5%
Hoa	0.86	1.1%
Thai	1.33	1.7%
Nung	0.86	1.1%
Tay	1.48	1.9%
Dao	0.62	0.8%
Hmong	0.79	1.0%
All Other	2.41	3.1%
Gia Rai	0.32	0.4%
Total	76.64	100.0%

The Hoa constitutes the sixth largest ethnic group in Vietnam. This group forms the traditional business community of Vietnam, often lives in urban areas, has broadly similar living standards to the Kinh and has a high rate of intermarriage with them (Baulch et al 2002). This section follows most of the literature, when it is not possible to calculate statistical data for individual ethnic minority groups, in classifying the Kinh and Hoa as the ethnic majority and all other groups as the ethnic minority.

Child Mortality Rates for Ethnic Minority and Majority Populations

The sample size of most household surveys is insufficient to calculate the infant or under-five mortality rate of individual ethnic groups. The 1997 DHS and 1997/1998 VLSS, for example, contain sample sizes of fewer than 6000 households. This means that for the Gia Rai ethnic group we would expect there to be approximately 24 households in the sample, which is an insufficient number to calculate a meaningful infant or under-five mortality rate.

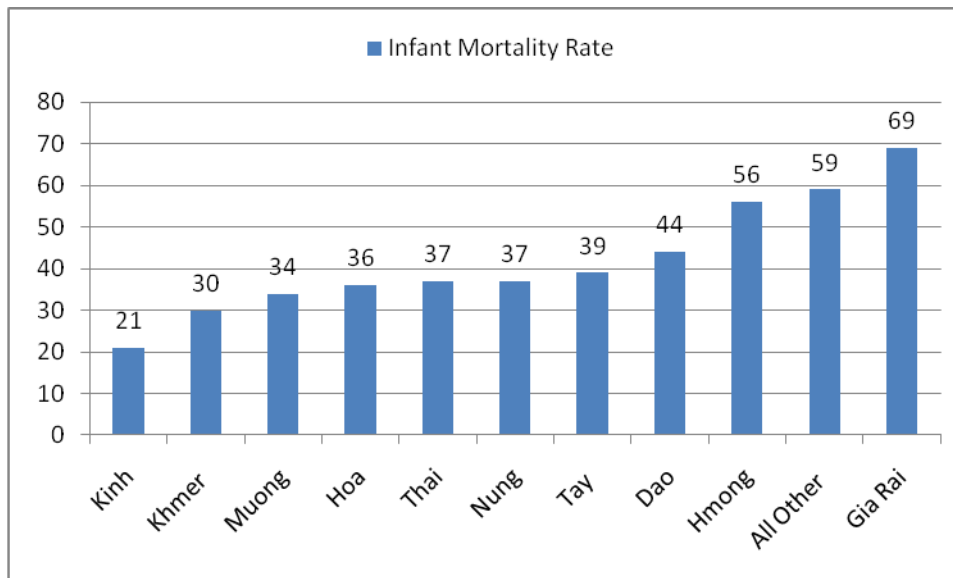
The health information system used by the Government of Vietnam in the 1990s and early 2000s did not record ethnic minorities as distinct groups. This means that it cannot be used to accurately assess trends in child mortality or its determinants for distinct ethnic groups (WHO 2003).

Because of the data constraints discussed above, we follow a two pronged approach when examining differences in child mortality between ethnic groups. First, we use data from the 1999 census to examine the 10 largest ethnic groups. Second, we use data from relevant household surveys to show child mortality rates for ethnic minorities and the ethnic majority (defined as Kinh and Hoa) ethnic groups.

Ethnic minority groups have higher mortality than the Kinh. Data from the 1999 census show that the Kinh had the lowest infant mortality rate. Some ethnic minority groups such as the

Dao, Hmong and Gia Rai had infant mortality rates that were more than twice as high as the Kinh (graph 13).

Graph 13: Infant Mortality Rates among Different Ethnic Groups (1999 Census)



There is also evidence from household surveys that the gap in child mortality rates between the ethnic minority and majority populations in Vietnam continued into the 2000s. Data from the 2002 DHS show an under-five mortality rate of 41 and 28 for the ethnic minority and majority populations respectively. The MICS of 2006 shows an under-five mortality rate of 35 and 25 among the ethnic minority and majority populations respectively.

All provinces in Vietnam have residents belonging to ethnic minority groups, but the Northern Uplands and Central Highlands have a higher concentration of residents from the ethnic minority populations. The Tay, Thai, Muong, Nung, Hmong, Dao, San Chay and San Diu are the main groups in the Northern Uplands. Infant mortality data are available for the Thai, Tay, Dao and Hmong ethnic groups. The population weighted infant mortality rate for these groups from the 1999 census is 42.3, which is significantly higher than for the Kinh majority (21) and slightly higher than the Northern Uplands region (40.9 DHS 2002). This suggests that high infant mortality among these ethnic minority groups may partly explain the high infant mortality rate in the Northern Uplands.

There are no nation-wide data on the epidemiology/medical causes of infant or under-five mortality by ethnic group. The higher rates of infant and under-five mortality seen among the ethnic groups in the Northern Uplands, however, suggests that they may not have progressed as far along the epidemiological transition as other groups in Vietnam and that the majority of under-five mortality will still be caused by infectious diseases. This point is supported by a study which found that in four poor Hmong and Dao ethnic minority communities in the Northern Uplands diarrhoea, ARI, pneumonia, bronchitis and dysentery accounted for the majority of morbidity among children under-five (Nguyen 2006).

Causes of Higher Child Mortality among Ethnic Minority Groups

Poverty, mother's education, water supply and sanitation and health outputs might explain the higher rate of child mortality among the ethnic minority population in Vietnam. This section examines each of these determinants in turn.

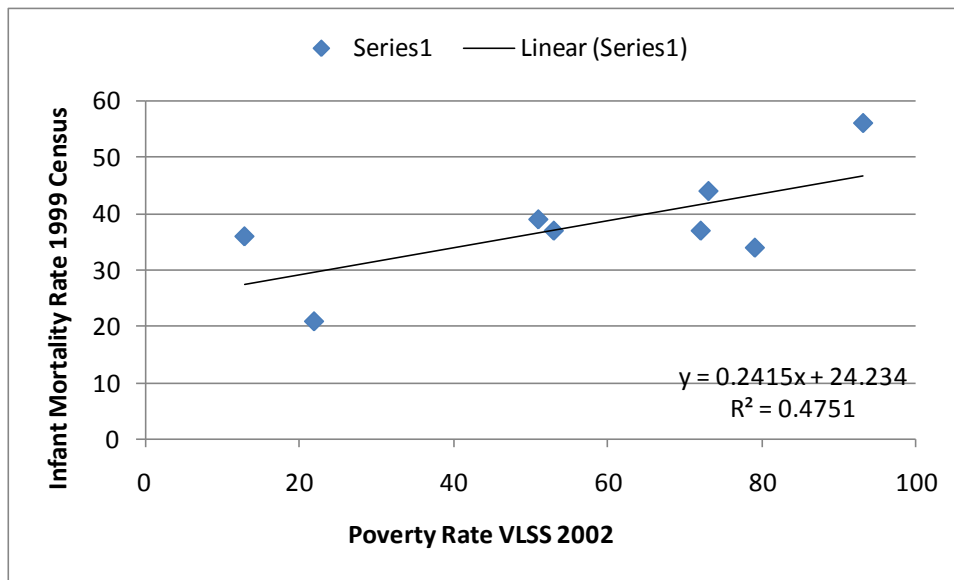
Over the 1990s and early 2000s economic development contributed to increases in family income and reductions in poverty for many families in Vietnam. Many ethnic minority families benefited from these economic developments. Poverty rates have declined steadily for ethnic minorities living in valleys and lowlands and engaging in paddy cultivation (WB 2008). And even ethnic minorities located in the Central Highlands and Northern Uplands have seen poverty decline. According to VLSS data ethnic minorities in the central highlands saw their poverty rate decline from 92% in 1993 to 70% in 2006; while for those in the Northern Uplands poverty declined from 89% to 59% over the same period. These are steep declines in poverty, but both the overall ethnic majority population (Kinh and Hoa) and the ethnic majority population in the Northern Uplands and Central Highlands saw much faster declines in poverty. The poverty rate among the ethnic majority in the Central Highlands declined from 45% to 9% between 1993 and 2006; while the poverty rate of the ethnic majority in Northern Uplands decreased from 69% to 9% (WB 2008). So the ethnic minority population was still much poorer than the ethnic majority population in the mid-2000s. Baulch et al (2002) and ADB (2002) also argue that the gap between the majority of the population and poorer ethnic minority groups widened throughout the 1990s.

The next graph further investigates the relationship between poverty and infant mortality across different ethnic groups using 1999 census and VLSS data. The data show that there is a strong relationship between these two variables; with a 1% reduction in the poverty rate leading to a 0.24 reduction in the infant mortality rate and this coefficient being significant at the 10% level. Part of the difference in infant mortality across ethnic groups may thus be explained by differences in poverty.

These results should, however, be interpreted with a degree of caution. The poverty rates on ethnic minority groups were taken from the VLSS 2002 survey because the sample size of the earlier VLSS was too small to calculate poverty rates for individual ethnic groups. This means that we are explaining infant mortality in 1999 using poverty data from 2002. This is problematic as low family income in 2002 can clearly not explain infant mortality three years earlier. We would, however, expect there to be a strong correlation between the poverty rates of different ethnic groups over time and therefore poverty in 2002 may be a reasonable proxy for the poverty of ethnic groups three years earlier.

A further problem with our analysis is that we have not controlled for other determinants of child mortality. Richer ethnic minority groups may well also be those that live in areas with better health facilities and access to clean drinking water. This means that the poverty rate may be acting as a proxy for the overall level of development and access to health services of ethnic groups and may not just reflect the relationship between family income and infant mortality. The causes of poverty among ethnic minority groups are complex and much debated and a detailed examination of them is beyond the remit of this thesis.

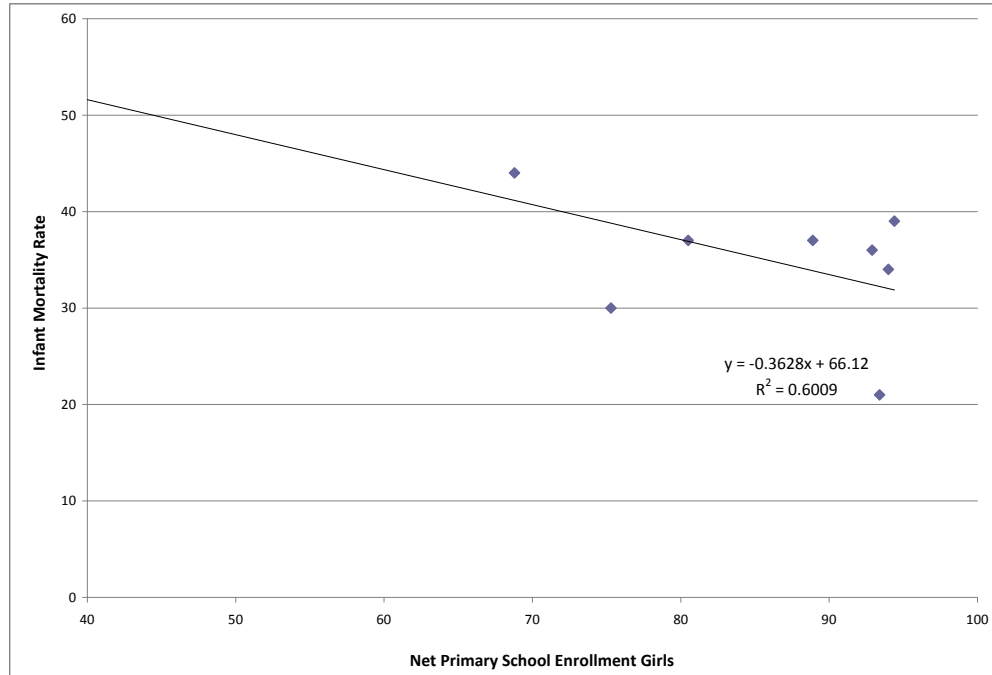
Graph 14: Poverty and Infant Mortality for Ethnic Groups



The education level of mothers can also reduce infant mortality because it may give them more control over household resources and increase children's consumption. In addition, educated mothers may demand and receive better access to healthcare and have superior knowledge of specific health interventions. Data on the education level of mothers by ethnic minority group are not available for the relevant period and our analysis thus uses net primary school enrolment of girls by ethnic group as a proxy.

There appears to be a strong relationship between primary school enrolment and infant mortality in 1999. Ethnic groups with high primary school enrolment have lower infant mortality; with a 1% increase in the net primary school enrolment of girls leading to a 0.36 unit reduction in infant mortality and this coefficient being significant at the 5% level. These results should, however, be interpreted cautiously as we have no direct data on mothers' education, have not controlled for other variables and have limited data points (see graph 15).

Graph 15: Primary School Enrolment and Infant Mortality by Ethnic Group



Other studies also directly link mothers' education to high child mortality among ethnic minority groups. Nguyen (2006) found that among four poor Hmong ethnic communities in the Northern Uplands many mothers lacked basic knowledge of health interventions. This study found that more than 90% of mothers believed that diarrhoea, a leading cause of under-five mortality, occurred naturally in children and was associated with their growth and development. Partly due to this belief use of Oral Rehydration Therapy was low. Many women also lacked knowledge of reproductive health practices including contraception and this contributed to poor birth spacing and possibly higher under-five mortality. Teerawichitchainan and Philips (2007) found that in both ethnic minority and ethnic majority households, educated mothers were more likely to report their child as sick and seek medical care. There is, thus, supporting evidence that lower education levels among ethnic minority mothers may increase child mortality.

Unclean water and poor sanitation can contribute to a worse disease environment and thus higher infant and under-five mortality. Statistical evidence from the 2004 VLSS shows that 82% of the Kinh and Hoa Chinese had access to an improved water source compared to just 37% of the ethnic minority population. ADB (2001) argue that access to clean water was still very low among ethnic minority groups in remote and mountainous areas in 2000 and that water supply become more problematic during the raining season.

Nguyen (2006) provides a vivid description of the problems with water supply and sanitation among four poor ethnic minority communities in the Northern Uplands. Most members of these communities reported that access to clean drinking water was a serious problem and that water tanks often ran out in the dry season meaning they had to spend considerable time searching for drinking water. Most families drank un-boiled water. Water tanks, meanwhile, were often left open and exposed to mosquitoes, flies and other disease-bearing insects. Cattle and other live stock were often kept in or close to houses. Nearly 90% of households

did not have access to fixed latrines and people and livestock often defecated near to water sources risking contamination. The author concludes that there was much evidence that poor sanitary and environmental conditions and a lack of clean water had contributed to an environment in which animal and human disease could quickly spread.

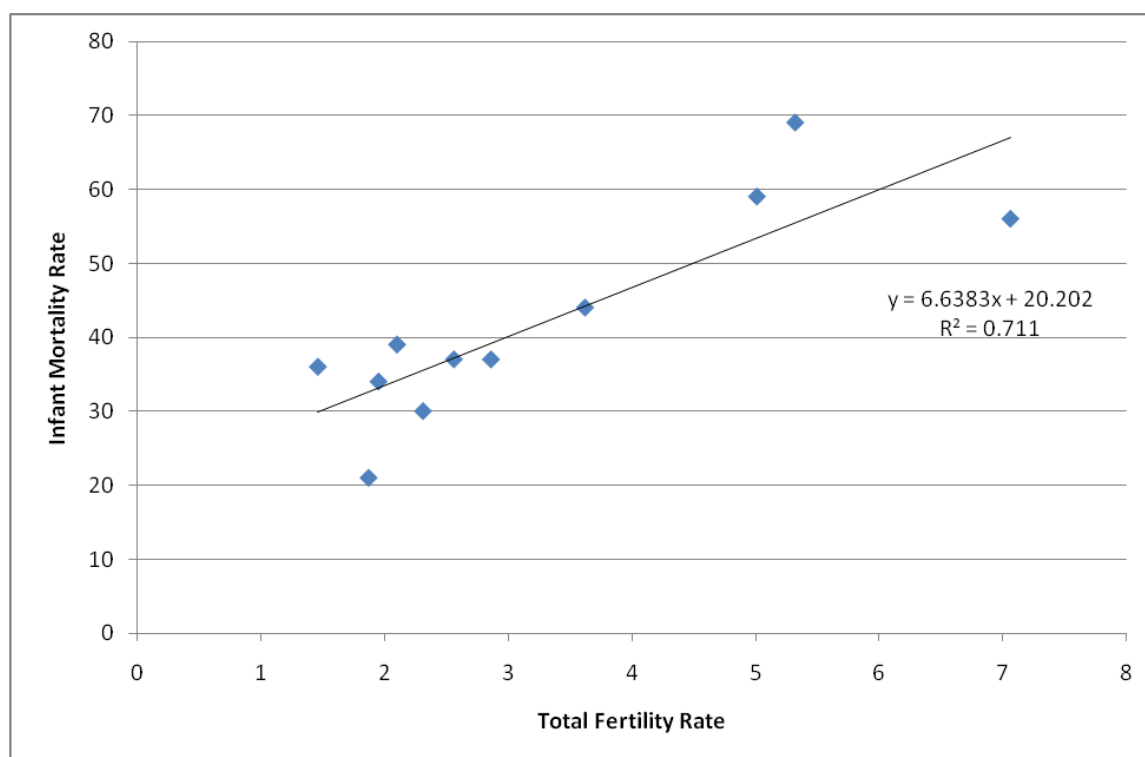
Child immunizations can significantly reduce under-five mortality. Official government statistics show that rates of vaccinations of over 90% for tuberculosis, polio, diphtheria, pertussis, tetanus and measles had already been achieved in Vietnam by the early 2000s (WHO 2003). Government statistics also show that there was only a small gap of less than 6% in vaccination rates between districts with high and low ethnic minority populations and that even the 10 provinces with the largest ethnic minority populations had vaccination rates of over 90%. WHO (2003) argue that vaccination programmes among ethnic minority groups have succeeded in both the Northern Mountains and Central Highlands.

Official figures on child immunizations may, however, significantly over estimate real coverage because the percentages are calculated in relation to preset targets (based on birth registrations in the present year) and not in relation to the total population of children (WHO 2003). Vaccination figures from household surveys show a lower overall level of vaccination and a more significant gap between the ethnic minority and majority populations. Data from the 2006 MICS show that 72.2% of ethnic majority children had received a full set of vaccinations against tuberculosis (BCG), diphtheria, tetanus and pertussis (DTP), polio and measles, mumps and rubella (MMR), but that just 35.9% of the ethnic minority population had received this full set of vaccinations. This gap in vaccination rates is arguably large enough to explain some of the variation in under-five mortality between the ethnic minority and majority populations.

Thang et al (2007) examine immunization coverage of children in Vietnam. This paper argues that immunization rates were significantly lower among ethnic minority children. Using a logistic regression analysis this paper shows that even after controlling for a mother's education level and household poverty, ethnicity was still a significant determinant of immunization coverage. The paper concludes that government programmes to increase immunization coverage should concentrate on poor ethnic minority communities.

Access to and use of contraception, through birth spacing and age of the mother at their child's birth, can be significant determinants of infant mortality rates. Ethnic minority groups in Vietnam generally have higher fertility rates, younger mother's age at birth and smaller birth intervals than the ethnic majority (WHO 2003). The next graph shows, using the 1999 census, that there is a strong statistical association between the total fertility rate and infant mortality for different ethnic groups. Specifically a one unit increase in the total fertility rate is associated with a 6.6 unit increase in infant mortality across ethnic groups and this coefficient is significant at the 1% level.

Graph 16: Total Fertility Rate and Infant Mortality across Ethnic Groups



The extent to which these differences in fertility are due to a lack of access to government family planning programmes and the extent to which they are due to a deliberate wish among ethnic minorities to have more children is difficult to determine. The MICS 2006 shows that the rate of use of modern contraceptives among the ethnic minority population is approximately 10% lower than among the majority population. This does not at first sight appear to be a large enough gap to explain the much higher fertility rate among ethnic minorities. These data are, however, of limited use because there are large differences in fertility across individual ethnic minority groups and because 1999 to 2006 saw a rapid decline in total fertility in Vietnam.

The general consensus in the literature is that the higher rate of fertility among ethnic minority groups is mainly because of economic circumstances and cultural factors meaning these groups wish to have more children. WHO (2003) argue that family planning remains a sensitive issue among ethnic minority groups and that there is evidence that the Hmong ethnic group rarely use IUDs even when they are available. It has also been argued that the government has been more flexible in the implementation of its two child policy among ethnic minority groups (WHO 2003). We tentatively conclude that a lack of access to contraception is not the principal reason behind higher fertility and poorer birth spacing among ethnic minority groups.

This section has examined the causes of higher child mortality among ethnic groups. Poverty, mother's education, access to water and sanitation, child vaccination rates and high fertility have all contributed to higher infant and under-five mortality among the ethnic minority population.

Health Policy and Systems with Relation to Ethnic Minority Groups

The government has a stated commitment to improving the well-being of the ethnic minority population. The 1992 constitution acknowledges the position, rights and obligations of all ethnic minority groups. The Government has also established the Programme for Socioeconomic Development in Communities faced with Extreme Difficulties in Mountainous Regions. This programme supported many ethnic minority communities in the early 2000s and has resulted in the construction of district hospitals, commune health centres and safe water supply systems in ethnic minority areas (WHO 2003 and WB 2008).

Vietnam has a dense health system which benefits ethnic minority communities. Nearly every commune has a CHC. There is also evidence that the number of hospital beds and CHC per capita is higher in ethnic minority areas than the national average and improved throughout the 1990s and early 2000s (WHO 2003 and WB 2001). Anecdotes of ethnic minority groups having to walk for days to reach a CHC are mostly a thing of the past (WHO 2003). Despite this, for a variety of reasons (mountainous areas, lack of transport and poorer roads) access to healthcare may still be more difficult for ethnic minority groups.

The Government liberalized the health system in the late 1980s and this included the introduction of user fees. Theoretically the introduction of users fees should have had a limited impact on under-five mortality because child health services were officially provided free of charge. Yet the reality is that a whole range of unofficial charges were also levied by hospitals and CHC (Segall et al 2002). The introduction of user fees may have been disadvantageous to ethnic minorities relative to the ethnic majority population for two reasons. First, ethnic minority groups are poorer than the majority of the population and fixed user fees represent a higher proportion of the poor's income and may therefore discourage them from seeking medical care. Segall et al (2002) provide some evidence that user fees may have reduced utilization of health services among the poor. Second, CHC and district hospitals in poorer ethnic minority areas may not be able to collect as much income from unofficial fees as health facilities in rich areas. This probably led to greater inequality in the quality of healthcare.

The limited availability of well-qualified staff who can speak the relevant local ethnic minority language may also hinder healthcare in ethnic minority areas (WHO 2003). Ethnic minorities, especially women, are under-represented in the healthcare professions. The waiving of user fees in some poor areas and the presumably lower unofficial fees also provides an incentive for better qualified health professionals to work in richer areas and avoid poor ethnic minority areas.

Overall ethnic minorities benefit from Vietnam's dense health structure, from exemptions from user fees and from specific programmes. There is, however, still evidence that the quality of the health system and healthcare is lower in ethnic minority areas and that the liberalization of the health system may have widened this gap.

Conclusion

Ethnic minorities have higher infant and under-five mortality than the ethnic majority population. Our analytical framework, empirical evidence and other studies suggest that this is because ethnic minority groups are poorer, have lower levels of female education, worse access to clean water and sanitation, higher fertility and worse access to high quality health

services. Given the lack of data on child mortality and its determinants among individual ethnic minority groups, it is difficult to determine the relative importance of these factors.

The Epidemiology of Child Mortality in Vietnam

This section examines the epidemiology of child mortality. We also discuss whether the changes in the epidemiology and distribution of child deaths could have been caused by social policies.

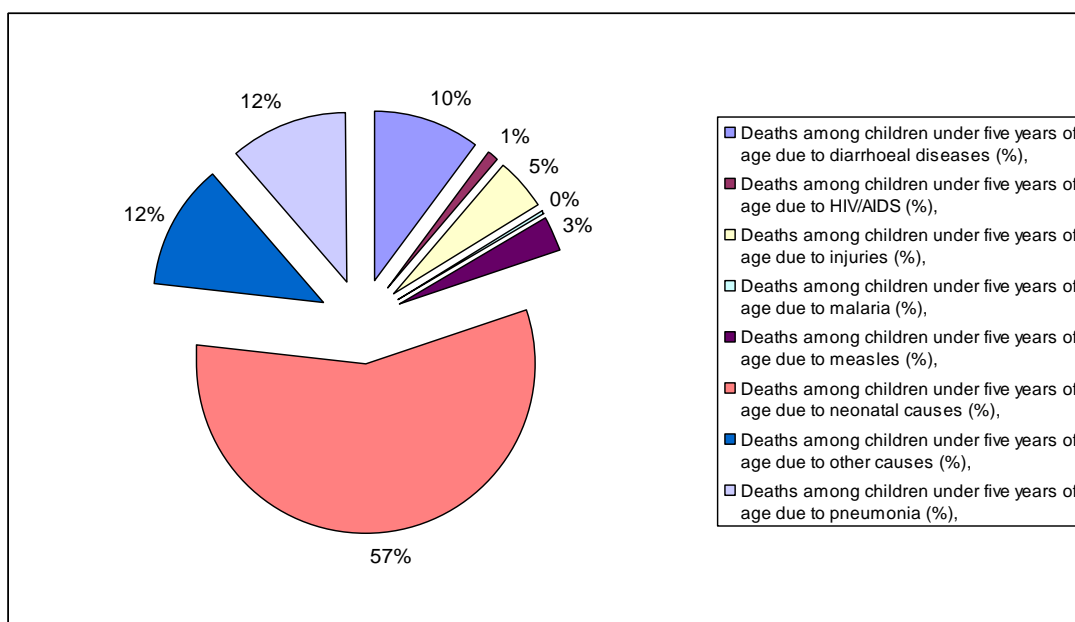
Declining under-five mortality in Vietnam has been accompanied by changes in the epidemiological causes of neonatal and child deaths. The total number and percentage of deaths caused by traditional communicable diseases among children and neonates have declined over the last 20 years. Polio was eradicated in 2002, neonatal and maternal tetanus were eliminated in 2005 and the incidence of measles has plummeted by 95% since 1990. This has meant that the proportion of child deaths caused by accidents has increased.

Graph 17 shows the percentage of under-five mortality due to different causes in 2000. It demonstrates that relatively few children in Vietnam die from malaria, HIV/AIDS, measles and diarrheal diseases. If effective government policies, and not just increasing family income, were responsible for reductions in deaths from these causes then this would provide strong evidence for our argument that effective social policies in Vietnam have contributed to the country's low and falling rates of under-five mortality. We discuss government policies for combating these diseases in greater detail in the next section.

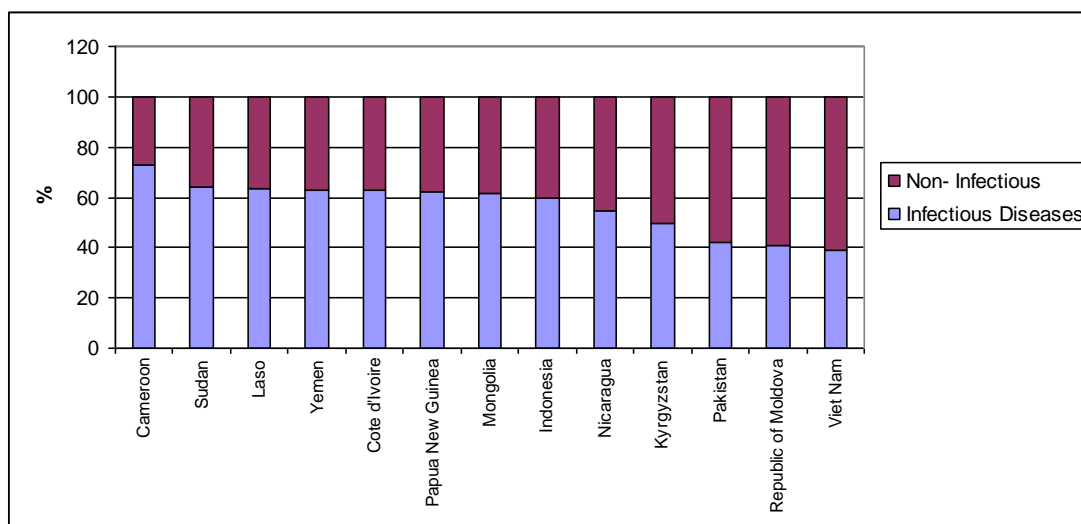
Vietnam's epidemiological profile of under-five mortality also differs from that of other developing countries. Graph 18 shows the percentage of under-five mortality caused by infectious and non-infectious diseases in Vietnam and other countries with similar GDP per capita. It clearly shows that in Vietnam, a much smaller percentage of under-five mortality occurs due to infectious diseases.

Graph 19 burrows deeper into the data to show the epidemiological causes of under-five mortality in Vietnam and our comparison countries. From this graph, we can observe that a smaller proportion of children in Vietnam die from pneumonia, measles, malaria and diarrhoeal causes than in our comparison countries. It is because of the low incidence of these infectious diseases that Vietnam has lower under-five mortality than countries with a similar level of wealth. If social policy has effectively targeted these diseases then this observation would provide strong evidence for our general argument that the reason Vietnam has low under-five mortality for its structural factors is because of the effectiveness of its social policies. The following sections of this chapter specifically investigate government policies for reducing these infectious diseases.

Graph 17: Under-five Mortality by Epidemiological Cause in 2000

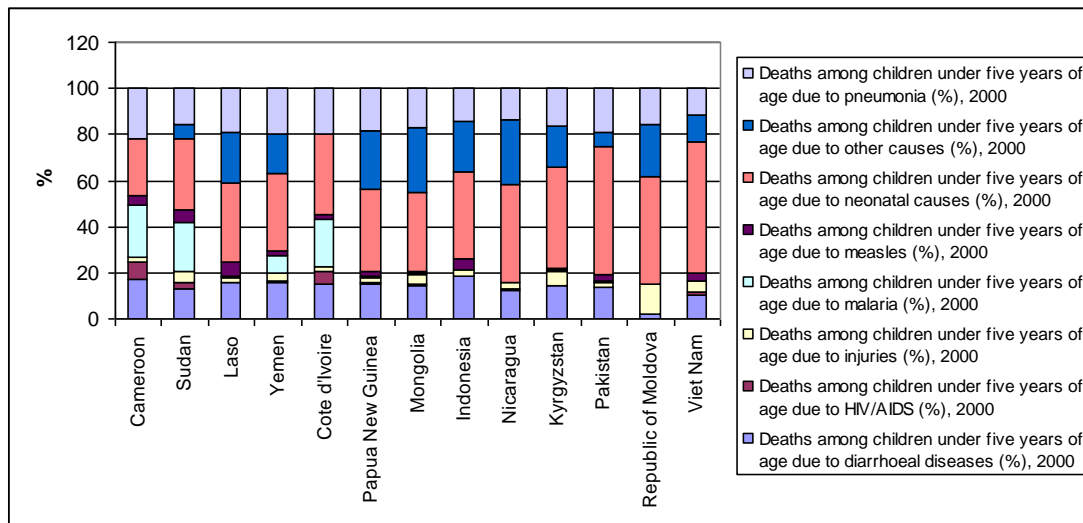


Graph 18: Infectious and Non-infectious Causes of Under-five Mortality in Selected Countries in 2000⁴⁹



⁴⁹ All countries selected had a per capita GDP of between 75% and 125% of Vietnam's in 2005.

Graph 19: Epidemiological Causes of Under-five Mortality in Different Countries



Conclusion

This section discussed the distribution of child mortality in Vietnam by region, family expenditure and ethnicity. It also discussed trends in child mortality in Vietnam over time and the epidemiology of child mortality in Vietnam. This discussion principally used data from the DHS, VLSS and MICS undertaken in Vietnam.

Inequality in child mortality by income has increased in Vietnam and is now as stratified as in many other developing countries. The outputs from relevant social policies are also now as stratified by income in Vietnam as in many other developing countries. Egalitarian social policies cannot, through diminishing marginal returns, explain why Vietnam has low child mortality for its structural factors.

There is significant variation in child mortality across regions. Differing rates of poverty, vaccinations and other health outputs between regions may explain this variation.

The ethnic minority population has higher under-five mortality than the ethnic majority in Vietnam. This is probably because ethnic minority groups are poorer, have lower levels of education, worse access to clean water and sanitation, higher fertility and worse access to high quality health services.

Vietnam saw sharply declining rates of child mortality in the 1990s and early 2000s. Based on current trends it is one of the few countries on course to achieve the fourth MDG. This makes it an interesting case study. It also provides some supporting evidence for our argument that social policies may have contributed to declining child mortality in Vietnam.

The number of child deaths due to infectious diseases such as pneumonia, measles, malaria and diarrhoea has fallen in Vietnam and is now lower than in countries with a similar GDP. Social policies' contribution to reducing child mortality from infectious diseases is further discussed in the next part.

5.4 Social Policy

This section examines whether social policy can explain why Vietnam has low child mortality for its structural factors. By social policy we mean government policies in the areas of family planning, female education, health and water supply and sanitation. Our earlier analytical framework showed that only public policies in these areas are likely to have a substantial impact on child mortality.

This analysis examines social policies until approximately 2005. Social policies after this date are not examined because the last child mortality data point in our regression was from 2006. Social policies that the government implemented after 2006 cannot explain Vietnam's low mortality for its structural factors in our original regression. Indeed as many social policies are likely to affect mortality with a substantial lag and our regression includes data from as early as 1989 much of the discussion in this chapter concerns social policies in the 1970s, 1980s, 1990s and early 2000s.

Family Planning

Causal Pathways between Family Planning and Child Mortality

Effective family planning can reduce child mortality. Lower fertility principally reduces child mortality by increasing the interval between births. There is substantial evidence from both rich and poor countries that conceptions occurring within 18 months of a previous live birth suffer a greater risk of foetal death, low birth weight, premature birth and small size and weight for gestational age (Conde-Agudelo et al 2006).

The potential impact of effective family planning and better birth spacing on child mortality is also significant. Recent evidence suggests that 9% of worldwide under-five mortality could be averted through the elimination of birth intervals of less than two years. Family planning is the most obvious, and according to recent evidence, one of the most cost effective ways of achieving this goal (Cleland et al 2006).

Reduced fertility may also improve child mortality through a second more complex, long term and controversial pathway. Dramatic reproductive change – the shift from many to a few births per woman – has in many countries been an important step towards gender equality; with falling mortality in most developing countries being associated with increased employment opportunities outside the home (UN 1995). This may in turn increase women's power, and thus through the mechanisms outlined in our analytical framework increase survival rates among children.

Evidence that Family Planning can and does Reduce Mortality

The causal pathways between reduced fertility and child mortality do not necessarily mean that family planning policy helps explain why Vietnam has low child mortality for its structural factors. For this to be the case, we need to demonstrate that family planning policy can reduce birth rates independently of economic growth and the overall modernization process, that Vietnam has low fertility compared to other countries and that Vietnam has an effective family planning policy.

Becker (1968) argued that poor couples rationally choose to have large families because they provide a supply of cheap labour and security in old age. This paper also points out that historically in Europe fertility fell sharply between 1800 and 1930 with little government support and in the absence of effective contraceptive methods.

Yet even if it is true that couples rationally choose to have large families, this does not mean that family planning programmes are bound to be ineffective. The reason is that many such programmes go beyond the provision of contraceptives and actually change the costs and benefits to having large families through fines and fiscal incentives. Family planning programmes can in short change the demand for children, and lead to rational people choosing to have fewer children. While the impact of lower fertility on economic growth may be complex and poorly understood; it directly and undeniably leads to lower child mortality. There is also much empirical evidence showing that family planning programmes lead to reduced birth rates.

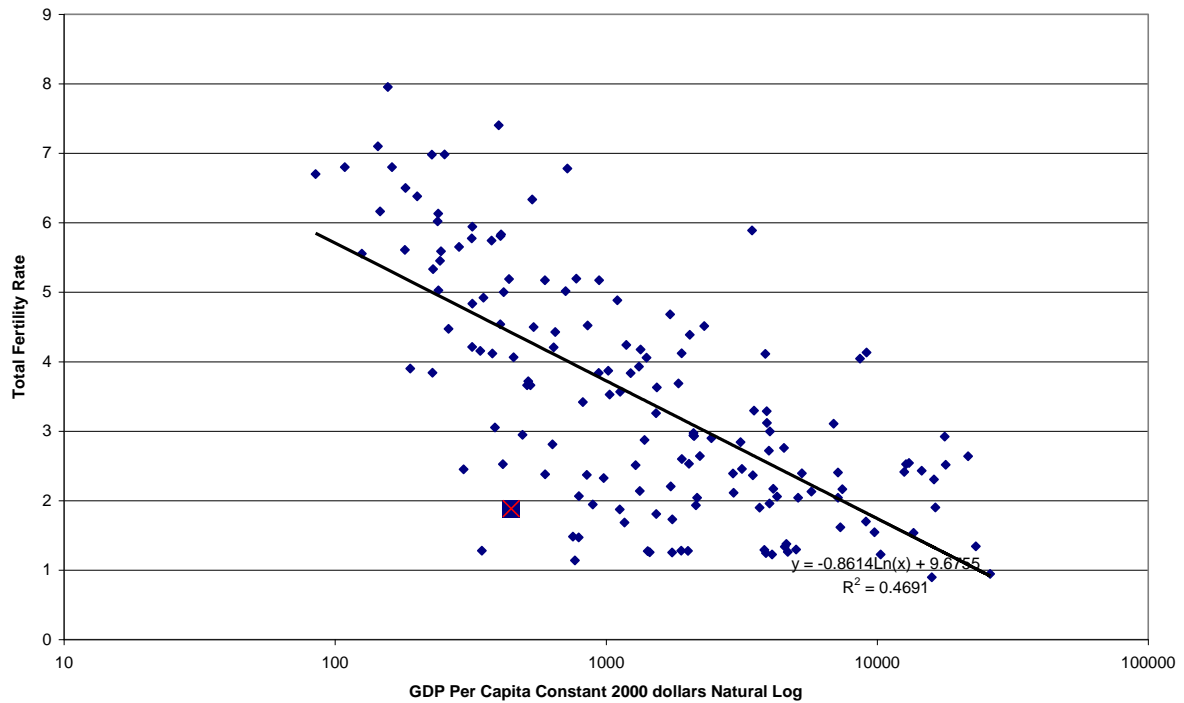
Reductions in fertility in almost all poor countries have happened in the presence of comprehensive family planning programmes (Caldwell et al 2002 and Potts 1997). There is also strong empirical evidence that family planning programmes can increase, and on occasion initiate, declines in fertility in both middle-income and poor countries (Phillips and Ross 1992).

There is a strong argument that if Vietnam had low fertility this could explain its low child mortality for its structural factors. Lower fertility could, in turn, be caused by government family planning programmes and not the general modernization process.

Vietnam's Fertility over Time and Compared to Other Countries and its Correlation with Mortality

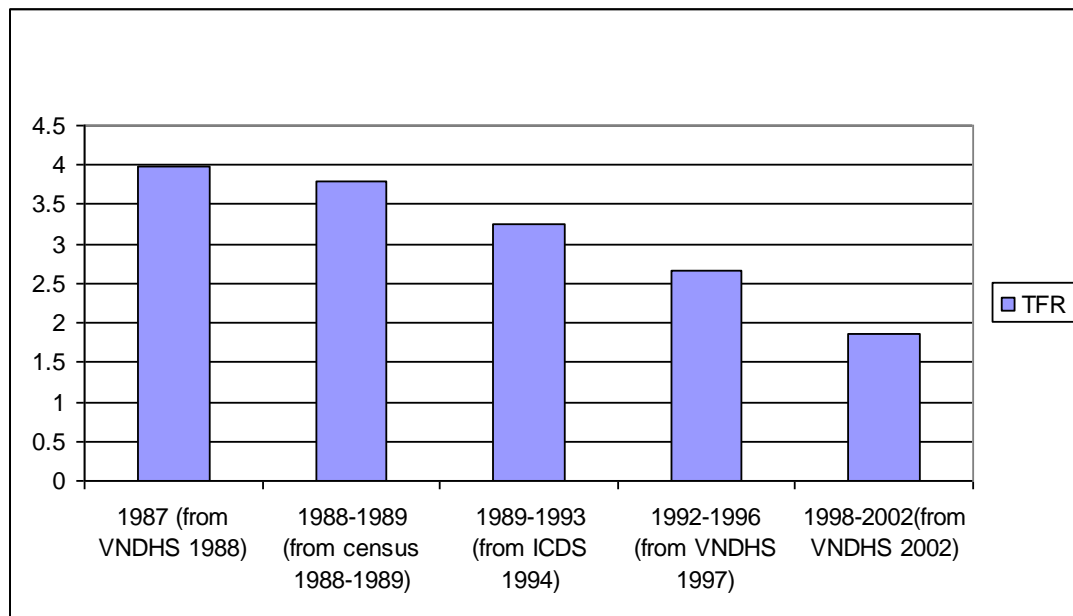
There is strong evidence that Vietnam has lower fertility than comparable countries. Graph 20 plots the total fertility rate and income of different countries. Vietnam is clearly an outlier in this graph (see cross in box); that is, it has low total fertility for its income.

Graph 20: Total Fertility Rates and Income (Average 2000 to 2004)



In addition, Vietnam has experienced sharply declining Total Fertility Rates (TFR). Fertility has declined from four children per woman in 1987 to 1.9 in 2002. This is an extremely steep decline in fertility. The rate of decline in fertility has increased in recent years; with the 0.8 decline in TFR from 1997 to 2002 representing a 30% decline over a five year period - a remarkable decline in fertility given that Vietnam already had low fertility in 1997. The next graph shows TFR from different surveys in Vietnam.

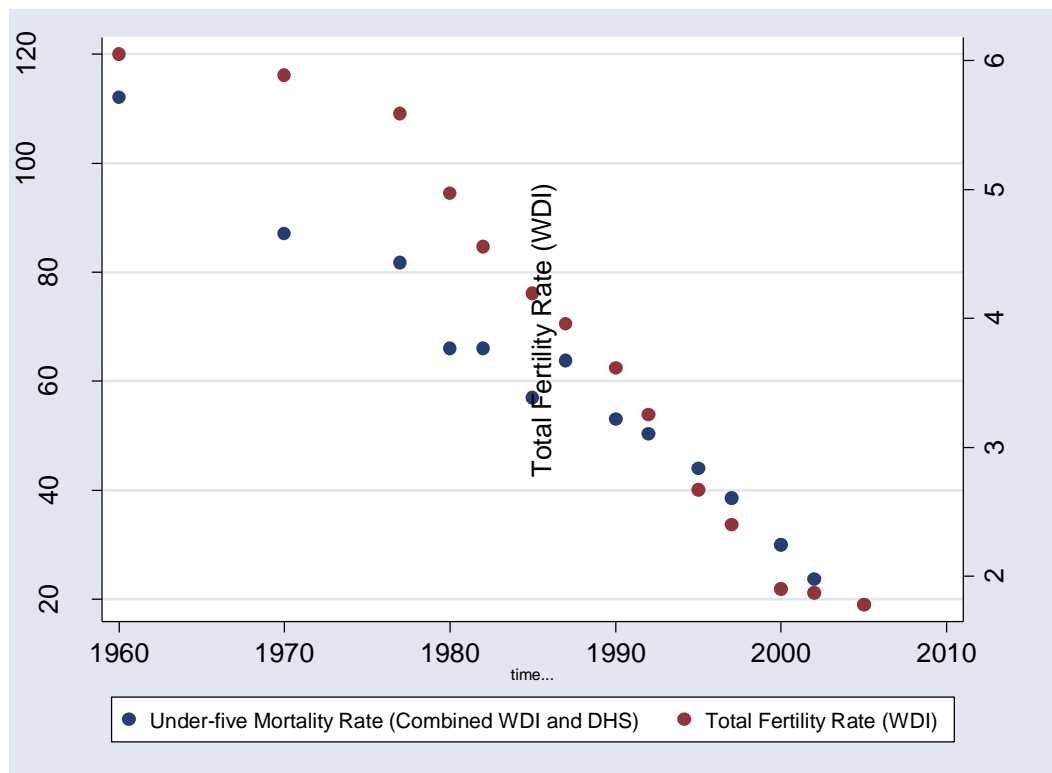
Graph 21: Total Fertility Rate in Vietnam from Different Surveys



The annual rate of decline of 6.4% in Vietnam's TFR recorded in the 2002 DHS may be internationally unprecedented. Mboup and Saha (1998) examine TFR declines in 10 countries with DHS data and find that that sharpest rate of annual fertility decline was 5.2% and occurred in Kenya. Rutstein (2003) calculates the rate of fertility decline in 21 countries (not including Vietnam) where DHS took place and concludes that Jordan experienced the highest rate of decline at 3.5%.

Declines in fertility in Vietnam are also correlated with sharp declines in under-five mortality (see graph 22). The decline in the TFR in Vietnam has also been associated with reproductive practices which are known to impact the health of neonates, infants and children. The age at which women have their first child has increased and adolescent fertility, which is known to increase child morbidity and mortality, has become increasingly rare (DHS 2002).

Graph 22: Total Fertility and Under-five Mortality in Vietnam over Time.



Birth intervals have also increased in Vietnam. More specifically, an analysis of data from the 2002 DHS demonstrates that 49% of second births occur four or more years after the previous birth and that fewer than 16% of births occur after an interval of less than 24 months. The median birth interval was 47 months in 2002, compared to 36 months in 1997 and the 32 months reported in 1994 (DHS 2002, DHS 1997 and ICDS). There is considerable research showing that short birth intervals, particularly those of less than 24 months, are harmful to babies' health.

There is therefore little doubt that low and sharply falling fertility is a contributory factor to Vietnam's low child mortality. But did the government have a family policy and if so was this more effective than in other developing countries?

Family Planning Policy in Vietnam

We examine government policy from the 1960s. The reason is that even though there may be a short lag between fertility and child mortality, there is a significant lag between family planning policy and TFR. For instance, policies implemented in the 1960s may legitimize the government's role in deciding how many children families have, making it easier for the government to implement effective family planning policies decades later. Public policy in earlier decades may also change cultural perceptions about family size; lowering fertility long after a particular family planning policy has been abandoned.

Vietnam's government has a longstanding and continuous commitment to family planning. The communist government of north Vietnam promoted a two or three family norm as early as 1963, but this policy was not initially enforced, promoted or adequately funded. During

this period there was no significant media campaign extolling the virtues of smaller families, contraceptives were not widely available, family planning services were only provided by the government in a few select locations and there were no fiscal penalties to having more than two or three children (Bryant 1988). Provinces repeatedly violated plan targets for total births and there were still fiscal incentives to couples having large families; with fourth, fifth and higher-parity children qualifying for rice rations and housing allowances, and their mothers qualifying for maternity leave (Jones 1982 and Werner 1984). During the 1970s contraceptives were made more widely available, with the family planning programme claiming a cumulative total of 600,000 IUD acceptors in north and south Vietnam combined by 1975. However, this figure cannot be used to estimate contraceptive prevalence rates since one woman can be counted as a new acceptor many times (Bryant 1988).

The 1980s saw a renewed and increased commitment to reducing fertility. Family planning policy throughout this period rested on three pillars – fiscal incentives and fines to promote ideal family sizes and reproductive behaviour, the promotion of the small family ideal through media campaigns, and the widespread distribution of contraceptives through the government's health network (Bryant 1988 and Goodkind 1995).

Norms for family size and reproductive behaviour were officially adopted in the 1988 Council of Ministers Decree on Population and Family Planning Policies. This document set a minimum age for first births of 22 years for women and 24 for men in urban areas or government employment and 19 years for women and 21 for men in other areas. It also placed a limit of two on the number of children for all families except for ethnic minorities and a minimum gap of three years between children. Although the enforcement and amount of fines varied by province, there is evidence that fines for non-compliance with fertility norms were common and large enough to affect birth rates. One survey reported that the majority of people in villages in northern Vietnam knew of at least one person who had been fined and that fines generally ranged from between one and three months' earnings (Goodkind 1995). First and second children also received preferential access to education, and additional land was only given to villagers for first and second births. Government workers have also faced fines for non-compliance with fertility norms. Tickets were often handed out to cadres outlining the appropriate timing for births and penalties for non-compliance included salary deductions and redundancy.

There was also a concerted media campaign to promote the ideal of the small family throughout Vietnam in late 1980s and 1990s. Billboards and posters promoting the one or two child family ideal became ubiquitous throughout Vietnam, with advertizements often depicting parents with a single daughter or daughter and son. These campaigns also emphasized the contribution that family planning could make to both family happiness and the national good (Goodkind 1995). The Ministry of Health has also distributed free contraceptives – mainly the IUD – through its network of health institutions which reach down to the commune level (Goodkin 1995).

It is also important to realize that while the one or two child policy was first implemented in the 1980s, and the majority of research on it undertaken in the 1990s, the Government of Vietnam is still committed to the goal of reducing fertility. The main mechanisms for promoting this goal – media campaigns, free contraceptives and fiscal incentives and fines – have remained in use. For instance in 2005, in response to newspaper articles falsely reporting a population surge, the government issued Political Bureau Resolution (Resolution

47/NQ-TW) on strengthening the implementation of population and family planning. This resolution reiterated the goal of achieving the replacement rate of fertility as soon as possible and the target of one or two child families. It also re-emphasized the policy of punishing party members and government officials who violated demographic norms.

Evidence on the Effectiveness of Vietnam's Family Planning Policy

There is also considerable evidence that family planning policy reduced birth rates. As we saw earlier, Vietnam has much lower, and has seen a much steeper decline in, birth rates than other countries with a similar level of income. This suggests that effective government policies have contributed to fertility decline. The decline in fertility rates also increased in the 1980s when the government started to implement the one or two child policy; although this was also a time of economic reform and increased economic growth in Vietnam – making it hard to distinguish the impact of specific policies from the general process of modernization.

In addition to this macro-economic evidence, most experts regard Vietnam's family planning programme to be one of the most effective in any developing country. An evaluation of 89 developing countries concluded that Vietnam had one of the six most effective family planning programmes (Ross and Stover 2000). Vietnam was one of the poorest countries in this group of six and had a much more effective family planning programme than that of other countries with a similar GDP.

Vietnam's family planning programme has also improved intermediate outcomes. The percentage of women who know of a modern method of contraception was already high in Vietnam at 94% in 1988 and continued to increase throughout the 1990s⁵⁰. Mass media campaigns and the promotion of modern contraceptives through the government's health system undoubtedly increased the population's knowledge of different contraceptives. The 2002 DHS reported that 88% of women surveyed had heard about the use and benefits of modern contraceptive methods on either radio or TV in the few months preceding the survey.

This knowledge of contraceptives also translated into their use. The 2002 DHS reported that nine out of 10 currently married women had used a family planning method. The IUD was the most popular method; with 65% of families having used this method. This report also found that 78.5% of women were currently using some method of family planning; with 56.7% of women using the IUD, making it the most common method. These figures also suggest that the government's family planning programme has been successful as the vast majority of contraceptives are supplied through public institutions. More specifically, only 11% of modern contraceptives are supplied by the private sector, with the largest suppliers of contraceptives being government-run CHC (45%) and hospitals (22%)⁵¹.

The unmet need for family planning in Vietnam is very low. More specifically, only 5% of currently married women have an unmet need for family planning while nearly 79% of women who are currently married are using family planning services. This implies that 94% of currently married women who are "demanding" family planning are currently using such a service (DHS 2002). There is little doubt that Vietnam has an effective family planning policy and that this has contributed to its low birth rate.

⁵⁰ DHS (2002). See: Graph 7 in appendix 12

⁵¹ DHS (2002). See: Graph 8 in appendix 12

In conclusion, Vietnam's low child mortality for its structural factors is in part explained by its low and sharply falling fertility rate. The low birth rate in Vietnam is in turn explained by the government's effective family planning policy. The main components of this family planning policy have been fiscal stimuli aimed at changing the number of children families rationally wish to have, mass media campaigns aimed at changing attitudes towards family size and the free provision of contraceptives through the government's health system. All three pillars of this policy have been highly successful.

Policies to Reduce Infectious Diseases

The Epidemiological Transition in Vietnam, and Links between Immunization and Child Mortality.

Vietnam has achieved the epidemiological transition. It has gone from being a country where a large number of children die, most of them from infectious diseases, to a situation where many fewer children die and a much larger proportion of child mortality occurs due to non-infectious diseases. The sharp falls in child mortality that we observed earlier, and Vietnam's outlying status compared to other countries, are partly explained by declines in under-five mortality due to infectious diseases. This section examines whether government policy caused this fall in child deaths from infectious diseases.

This section examines immunization policy in the 1980s, 1990s and early 2000. We do not examine government policy in earlier decades because there is only a small lag between immunization and mortality. The hospitals and CHC used to implement immunization policy may have been built in earlier decades, but these are examined later in this chapter in the health policy section.

Experts have long recognised that immunizations are a medically and economically effective way to reduce child deaths from infectious diseases such as tuberculosis, diphtheria, pertussis, tetanus, polio and measles. For malaria – an infectious disease that commonly kills children – there is no immunization but effective preventative methods such as spraying and bed nets exist. This chapter, therefore, evaluates public policies for promoting immunizations and malaria protection and treatment in Vietnam.

Vaccination Policies

The Government of Vietnam has a longstanding policy for vaccinating children. In 1981 the government implemented the expanded programme of immunization. This programme provided free vaccinations to children for tuberculosis, diphtheria, pertussis, tetanus, polio and measles. In 1988 the programme was expanded with new targets for eliminating neonatal tetanus (by immunizing pregnant women), and controlling measles. Vaccinations were also introduced for hepatitis B, Japanese encephalitis B, typhoid and cholera. In 1999 in recognition of the programme's uneven coverage supplementary immunization campaigns were introduced in certain underserved geographic areas.

A further campaign was introduced in 2002 and 2003 to provide further measles vaccines to children aged nine months to 10 years. The current programme on immunization aims to ensure that every region has an immunization rate in excess of 90% for all six basic childhood diseases. It also aims to defend the elimination of polio and neonatal tetanus; expand immunizations against Japanese encephalitis; increase coverage of immunizations for

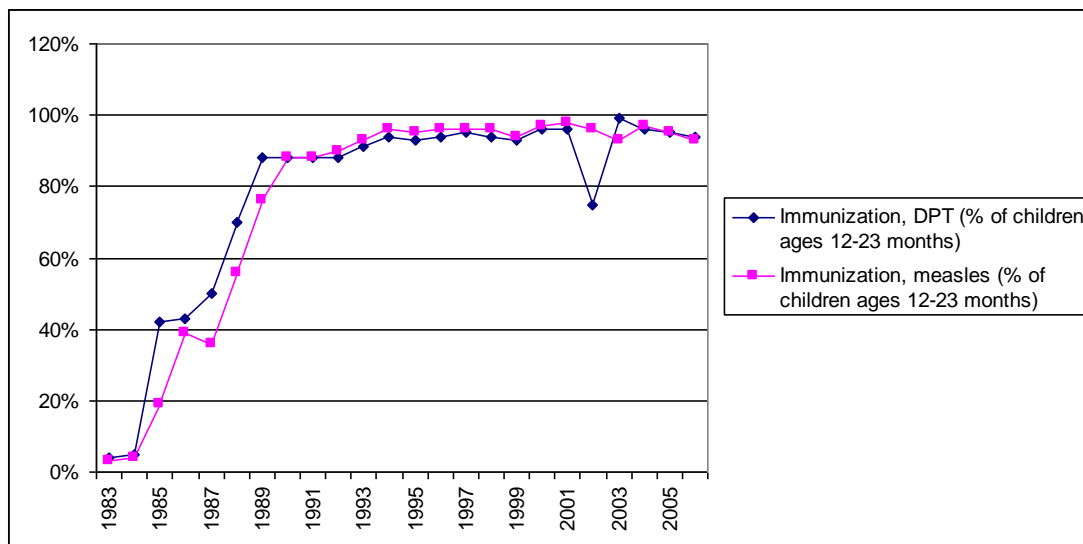
typhoid and cholera in endemic areas; and reduce the incidence of diphtheria and pertussis through booster shots of DPT (UNICEF 2011).

Success of the Government of Vietnam's Vaccination Policies

These vaccination programmes successfully improved immunization rates. By 1989 an immunization rate in excess of 80% had been achieved and by 1989 polio had been eliminated. Graph 23 shows that rates of immunization for the most common childhood infectious diseases continued to increase throughout the 1990s. Increased childhood immunization rates undoubtedly contributed to the sharp decline in under-five mortality in Vietnam.

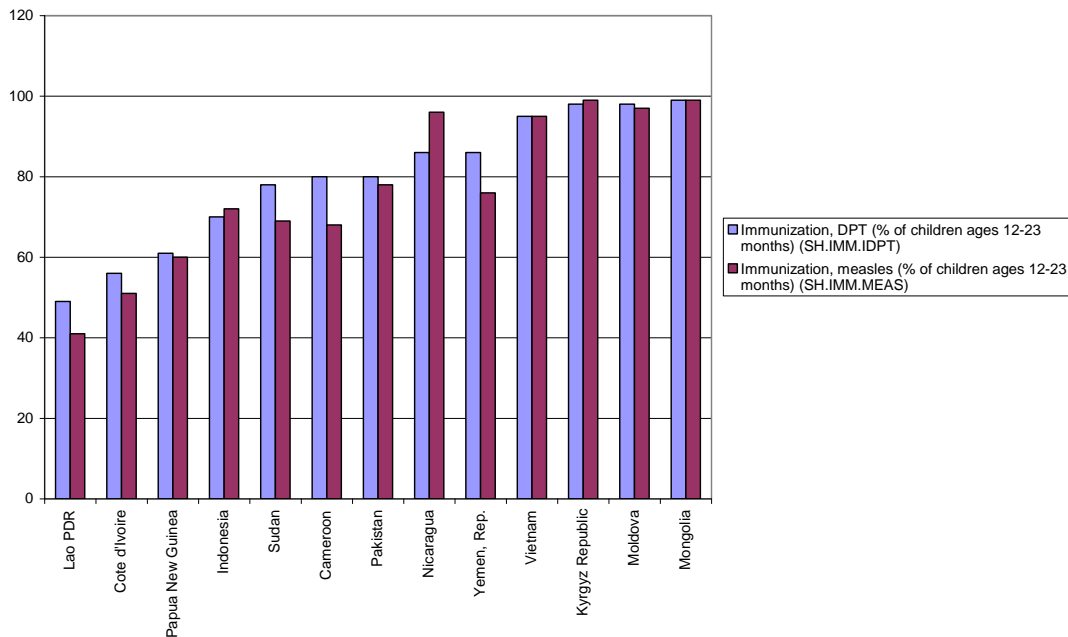
Immunization rates are also higher in Vietnam than in most other developing countries. Graph 24 compares rates of immunization for measles and DPT in Vietnam and a selection of other countries with a similar per capita GDP.

Graph 23: Vietnam Immunization Rates⁵²



⁵² Data are taken from the WDI Database. Original source of data is UNICEF and WHO.

Graph 24: Immunization Rate in Vietnam and Similar⁵³ Countries in 2005⁵⁴



There is little doubt that government policy increased immunization rates and reduced under-five mortality. We can attribute the success of this policy to a range of factors. First, the government was clearly committed to improving immunization rates over a long period of time – since at least 1980. The aims and programme goals remained remarkably consistent from the early 1980s until the present. Second, immunizations were provided free of charge; which encouraged a broad spectrum of the population to access this service. Third, as discussed in greater detail later in this chapter, Vietnam built an extremely dense health network, with commune health centres operating in most communes. These facilities provided immunization at a convenient location along with other important services such as family planning. This dense health system also allowed the government to effectively monitor immunization rates. Fourth, the government offered fiscal incentives to health workers; with additional payments being made to workers who actually administered the vaccines based on the number of fully immunized children.

The Government of Vietnam's Policy to Control Malaria and its Impact on Child Mortality

In many developing countries substantial numbers of children die from malaria. This was probably the case in Vietnam in the 1960s and 1970s, but in more recent times deaths from malaria have sharply declined in Vietnam (WHO 2003b). This section examines the role government policy played in reducing child mortality from malaria.

The government launched a malaria control programme in northern Vietnam in 1958, which was expanded to the rest of the country in 1976. This programme had some initial success in the north but from the early 1970s there was a resurgence in malaria. The failings of this

⁵³ All countries selected have a per capita GDP of between 75% and 125% of Vietnam's in 2005.

⁵⁴ Data are taken from WDI Database. Original source of data is UNICEF and WHO.

programme were mainly due to a shortage of financing leading to a shortage of anti-malaria drugs and insecticide. Migration after the Vietnam War may also have increased incidences of malaria (WHO 2003b).

The government implemented a new malaria strategy in 1991. The government moved away from mass DDT spraying and towards the free provision of drugs and mosquito nets, which themselves were re-sprayed twice a year for free. A new drug Artemisinin was also introduced during this period (WHO 2003b). The government also introduced an intense malaria education programme for village heads, commune health workers and cadres.

This new policy quickly improved intermediate outcomes. The population protected by impregnated bed nets increased from 300,000 in 1991 to over 10 million by 1997. The number of doses of Artemisinin quickly increased with four million doses being distributed between 1991 and 1998. These increases in bed nets and access to drugs contributed to the reduction in under-five mortality due to malaria (WHO 2003b).

There were four main reasons for the success of this programme. First, in Vietnam there was a large cadre of health workers who could be trained in malaria identification, treatment and prevention and closely monitor the use of bed nets. By 2003 nearly 80% of treatments were carried out at the community level. Second, the high levels of literacy in Vietnam made it easier to educate the population on the importance of using bed nets. Third, Vietnam was able to adapt international best practice to its own circumstances and hence reduce costs. For instance the programme invested money to domestically produce malaria drugs and only sprayed nets with half the recommended dose. Fourth, the programme consistently received high level political support, with the president chairing the National Conference on Malaria in 1992, to emphasise the importance of the new programme (WHO 2003b).

This section analyzed government policy for treating infectious childhood diseases. Government policy in Vietnam reduced deaths from infectious diseases in children more effectively than in many other countries. This partly explains why Vietnam has low child mortality for its structural factors.

Health Policy and Systems

This section discusses whether Vietnam's health policy and systems explain its low child mortality for its structural factors. The Government of Vietnam has, as discussed earlier, effective family planning, child immunization and malaria programmes. These were implemented through the general health system. The health policies and systems examined in this section can thus help to explain the effectiveness of immunization and fertility policy and their impact on child mortality.

The evolution, structure and policies of Vietnam's health system are complex and many studies have examined them in detail⁵⁵. Consequently, there is no value added to undertaking a detailed descriptive review of Vietnam's health system and we do not attempt such a review here. Rather we examine three seminal determinants that explain why Vietnam's health system and policies are, compared to other developing countries, extremely effective in improving child survival. The key determinants examined here are: the density of the health

⁵⁵ See: WB (2001), Sepehri et al (2004), Gabriele (2006) and WB (2008).

network, the quality of child healthcare and the extent to which the health system ensures equity and protection from the vagaries of the market.

The Development of Vietnam's Health System in the 1970s and 1980s and its Structure

Throughout the 1970s and early 1980s the government established a dense and arguably extremely effective health system. This was organized along a five tiered pyramid. The highest point in the pyramid is the Ministry of Health, which is the main national authority in the health sector. The Ministry oversees the manufacture and distribution of drugs, physician training and health research. It also bears ultimate responsibility for the provision of all preventative and curative health services in Vietnam (WB 2001 and WB 2008).

Provincial health bureaus make up the second tier of the system. These service a population of up to five million people and are funded from local government budgets. These bureaus are mainly responsible for the planning of health services and programmes. Each province has at least one general hospital, with 200 to 1000 beds and seven departments, namely – internal medicine, obstetrics, gynaecology, paediatrics, infectious diseases, traditional medicine and an emergency ward (WB 2001 and WB 2008).

The third tier of the pyramid consists of district health centres. These manage health services in each district and are in principle responsible for the management of all health activities at the grassroots level – although in some cases they only have direct control over personnel and salaries. Each district also has a general hospital which includes posts for hygiene, epidemiology and malariology and normally a unit for maternal and child health and family planning. The district hospitals also provide training for the staff of the grassroots institution of healthcare in Vietnam – the CHC (WB 2001 and WB 2008).

CHC make up the fourth tier of Vietnam's health system. These centres normally employ three to five staff and in 2006 a doctor worked in approximately 70% of them. CHC's principal task is to provide a range of basic health care services to the local population. Services provided by CHC include preventative care, family planning, attending births, immunization, the supply of basic drugs and overall health promotion. These centres are essential for the delivery of the government's national programmes including child immunization, child nutrition and family planning (WB 2001 and WB 2008).

Village health workers make up the bottom tier of Vietnam's health system. These workers are managed by CHC and are responsible for community outreach programmes and assisting in the provision of basic medical procedures such as immunizations. In 2006, approximately 80% of villages had such health workers.

Importance of Vietnam's Health System and its Role in Reducing Child Mortality

CHC have played a key role in improving rates of child survival by providing immunization and family planning services. There is much evidence that Vietnam had a denser and more effective network of CHC than other comparable countries. By 1997 Vietnam had over 9,806 CHC which served an average of approximately 7000 people. This compares favourably to Cambodia and Thailand (a richer country) where similar centres served 15,000 to 6,762 people respectively. The establishment of this dense network of CHC represents a remarkable achievement for a developing country. And the establishment of this network undoubtedly contributed to the rapid declines in under-five mortality seen in Vietnam (WB 2001). Other

indicators also suggest that Vietnam has a dense health network. Vietnam has a significantly higher number of hospital beds, physicians and skilled health personnel than most other countries with a similar GDP per capita⁵⁶.

Vietnam's health network also provides greater access to relatively high quality child healthcare than in comparable countries. A much higher percentage of births are attended by a trained health professional in Vietnam than in other countries with a similar GDP per capita⁵⁷. A WHO (1998) study concluded that a higher percentage of children in Vietnam are correctly assessed, treated and diagnosed for diarrhoea than in Malaysia, Bangladesh, Indonesia or Myanmar.

Prior to 1986 Vietnam's health sector was publicly funded and healthcare was available free of charge at the point of demand. Vietnam's health system may have been more effective than other countries' systems in this period because its government did not rely on the market to provide healthcare and instead ensured equitable access to a range of preventative and curative health services for children. The provision of healthcare in the market place will not lead to the minimization of child mortality for two main reasons. First, there are significant externalities in curing infectious diseases; for instance ensuring a child does not get polio benefits everybody who could potentially catch polio from that child and not just the individual who is actually immunized. And second, in many countries the poor may simply be unable to pay for measures which increase the survival chances of their children. This theoretical evidence has lead most commentators to conclude that it is essential for governments to take concerted action to ensure there is widespread and equitable access to basic healthcare for children (Bryce et al 2003). There is also no doubt that in Vietnam government spending created a dense and effective health system, which reduced child mortality.

Reforms to Vietnam's Health System from the Late 1980s

Reforms were implemented to liberalize Vietnam's economy in the mid-1980s. In 1989 market-oriented reforms were introduced to the health service itself. These reforms included the introduction of user fees, legalization of private medical practice and the regulation of the retail trade in drugs and medicine.

There is no doubt that in general these reforms lead to the health system becoming increasingly market-oriented; with public hospitals responding to market signals in the treatments they offered, the rich paying for and receiving a better standard of care in both the public and private systems and more drugs being directly purchased from private providers (WB 2001). There is also little doubt, as shown by the statistical analysis undertaken earlier, that rates of infant and under-five mortality rapidly declined in Vietnam throughout the 1990s, and in the early years of this century. A cursory analysis could then conclude that an increasingly market-oriented health system provided a higher standard of care to children, reducing child mortality.

This conclusion may be incorrect because increasing real family income contributed to declining child mortality in the 1990s: when health systems reform and real incomes increase it is difficult to determine which is driving changes in mortality. Our econometric analysis,

⁵⁶ Author's own calculation based on WDI data. See also graphs 9 and 10 in appendix 12.

⁵⁷ See: Graph 11 in appendix 12.

however, shows that Vietnam had low child mortality for its structural factors after it reformed healthcare. This suggests that Vietnam's reformed market-oriented health system may have been effective in reducing child mortality compared to other countries. It is also wrong to assume that all health services are equally subject to market discipline. A detailed examination of how the reforms affected those services most relevant to children is thus warranted.

Healthcare reform initially reduced the quantity and quality of care provided by CHC. The main reason was that these centres had traditionally been funded by agricultural work brigades, which began to disappear with the onset of the reform process. CHC lost their main source of funding; leading to a deterioration of their physical infrastructure and a reduction in the quantity and quality of drugs supplied to the local population (WB 2001). One study estimated that only 49% of CHC in rural areas had a functioning sterilizer and only 58% had a usable weighing scale for infants by the late 1980s (WB 1992). Inflation and the end of housing and education benefits had also substantially devalued the real remuneration of commune health workers by the late 1980s (Dung 1996). Salaries were also paid irregularly (Dung 1996). These problems led to a decline in the standard of healthcare provided by CHC and a reduction in the health workers in these centres from 58,665 in 1985 to 39,701 in 1990 (WB 2001).

Thus in the early 1990s, the system of CHC, which were the most important part of the health infrastructure for child healthcare, were nearing collapse (Sepehri et al 2004). The government responded by ensuring that from 1994 health workers were directly paid out of the provincial health budget. By 2006 about three quarters of the operating budget for CHC was paid for by the government (WB 2008). Funding is normally provided by local governments although disadvantaged communes' CHC are funded directly by central government. These reforms effectively saved the system of CHC from collapse and meant that many of the services most essential to the reduction of infant and child mortality rates did not face the vigour of the market to the same extent that other components of the health system did (WB 2008).

The introduction of user fees could also have substantially affected child healthcare. Proponents of user fees argue that they increase funding directly to service delivery units; encourage hospitals to provide those services which the public want; because demand is inelastic they do not substantially reduce the number of people seeking healthcare and they stop secondary and tertiary hospitals being overused. Opponents of user fees argue that they reduce access to health services; impoverish the poor; increase inequality and because many diseases have negative externalities, lead to a sub-optimal provision of healthcare.

Empirical evidence from Vietnam presents a mixed picture of the costs and benefits of user fees. There is a great deal of evidence that user fees represent a significant expenditure for poor households and that unforeseen medical emergencies can force them into debt or to sell productive assets (Segall et al 2002). Indeed it has been calculated that poverty would be 1.1% lower if there were no out of pocket health expenditures (WB 2008).

There is also evidence that user fees lead to the poor using health services less (Segall et al 2002). This may have contributed to the falling utilization of health services and increased inequality in access to healthcare and health outcomes in the 1990s. On the other hand, user fees directly increased the revenue of many health units and thus helped fund improved services, health infrastructure and health professionals' salaries (WB 2008). The decline in

the utilization of health services also started before user fees were introduced. Utilization began to increase in the late 1990s possibly because exemptions from fees and greater access to health insurance began to reduce user fees' impact on the poor (WB 2001 and WB 2008).

The direct impact of user fees on child health is difficult to measure. They should have had a very minor impact as child health services were meant to be provided free of charge. Families can claim this exemption by presenting their free healthcare cards, which provincial health department figures show have been distributed to 96% of children, or by presenting other identity papers including birth certificates and commune birth registration documents (WB 2008).

However, unofficial fees may well have been charged by CHC with one empirical study finding that 75% of the total costs of services in these centres were covered by unofficial user fees and another reporting that 72% collected unofficial fees to cover essential operating expenditures (Dunlop 1999 and WB 2008). Studies such as Segall et al (2002) and Sepehri et al (2004), using VLSS data, have examined the impact of user fees on access to healthcare, but these studies do not specifically investigate whether or not user fees affected the extent to which parents took their children to CHC or hospitals. So there is limited evidence on the impact of user fees on access to child healthcare. The high rates of immunizations and success of the vertical health programmes throughout the 1990s do, however, suggest that while unofficial user fees existed they were not high or widespread enough to seriously erode children's access to healthcare.

A second major component of the reform agenda was the liberalization of the market for pharmaceuticals. This reform succeeded in increasing the supply and reducing the cost of drugs. CHC, which were often chronically short of drugs in the 1980s, had adequate drugs supplies during the 1990s in Vietnam (WB 2001). The relative cost of drugs also fell throughout the 1990s (WB 2001). Yet the liberalization of the drugs market also increased the incidence of self-medication; leading to increased anti-biotic resistance in some diseases (Lonroth et al 2001 and WB 2001). Drugs also became a significant out of pocket expense especially for the poor and over-subscription occurred because hospitals, CHC and health professionals could supplement their income through drugs sales (Lonroth et al 2001). The relative impact of these positive and negative effects of drug liberalization on child health is difficult to quantify and indeed no epidemiological study has attempted to do so. The falling number of child deaths from infectious diseases would, nevertheless, appear to indicate that liberalization of the market for drugs did not have a substantial negative effect on infant and child mortality.

Concluding Comments on Vietnam's Health System

In conclusion, Vietnam's health system and policies help explain why it has low child mortality for its structural factors. The dense network of CHC was effectively used to implement immunization and family planning programmes that reduced infant and child mortality. There is also substantial evidence that this system was denser and more effective than primary health systems in other developing countries. In the late 1980s the government began to liberalize the health system in Vietnam. The impact of the reform process on child healthcare was, however, complex and did not simply involve child health services being subjected to the full vigour of the market. CHC were saved from collapse by the government's decision to directly fund some of their expenditure out of general revenue. Their funding was made more, and not less, dependent on revenue from general taxation

during the reform process. Furthermore vertical health programmes such as child immunization and family planning continued to be officially supplied free of charge. The general increase in funding received by hospitals and CHC from the fees they were charging for other services helped maintain and improve the health infrastructure and increase the remuneration of health professionals. So the reform process effectively led to payments for other health services subsidising child healthcare. Child health services, then, benefited from the liberalization of the health system in Vietnam.

Poverty Reduction, Health Insurance and Social Protection

The government reformed and liberalized the health system and the economy throughout the 1980s. This liberalization meant that income inequality increased; with some faring better in the market place than others. By the 1990s, Vietnam's health system was effectively a mixed system; with both private and public institutions providing services and public health centres and hospitals charging official and unofficial fees. The combined impact of these joint reforms was that inequality in peoples' ability to pay increased and that the extent to which the quality of child healthcare was determined by income increased. As discussed earlier, this contributed to increased inequality in child mortality in Vietnam throughout the 1990s; with child mortality decreasing faster for rich than poor families.

The Government of Vietnam has responded to these inequalities by introducing a range of poverty reduction programmes and subsidized health insurance. Social protection programmes which also have the potential to reduce income inequality and thus access to healthcare in a market system were also expanded. At first glance it might appear that government policies in these areas could help explain why Vietnam has low child mortality for its structural factors compared to other developing countries. Yet this is not the case. For a variety of methodological and practical reasons – health insurance, social protection and poverty reduction programmes cannot and do not explain why Vietnam is an outlier in our regression analysis.

Health insurance coverage cannot explain low child mortality in Vietnam because children are officially exempt from user fees. There are other significant cost barriers for gaining access to child healthcare: time taken off work, transport costs and unofficial fees may all reduce access to healthcare, especially among the poor. But health insurance does not provide for transport costs or income lost through work, and it is unlikely that unofficial fees would be waived for people simply because they were covered by health insurance or that health providers would attempt to be reimbursed for these through a formal scheme. Unofficial fees are, by their nature, not covered by official government policy or insurance. The coverage and effectiveness of health insurance is thus, mainly, irrelevant when attempting to explain why Vietnam has low child mortality for its structural factors.

Poverty reduction and social protection programmes potentially redistribute income to poor households. The redistribution of income to poor households would reduce child mortality if there were diminishing marginal returns between income and the effectiveness of healthcare. That is, if a rich family increasing its health expenditure on children from \$4000 to \$4050 only leads to a small decrease in the chance its child will die, while an increase in a poor family's expenditure on its children from \$100 to \$150 leads to a larger reduction in the chances of death.

Yet the effect of poverty reduction programmes and social protection on income inequality cannot be used to explain why Vietnam has low child mortality compared to other countries for its structural factors. The reason is that one of our structural factors, which we have previously controlled for and thus cannot use as an explanatory factor here, is income inequality. The exact measure we used to control for income inequality – the Gini coefficient – measures income ex-post social policy. That is, it measures income inequality after transfers from poverty reduction programmes and social protection. So our econometric model has already accounted for the “income redistribution” impact of these policies.

Poverty reduction programmes did also include components aimed at minimizing income based inequalities in access to education. And this is relevant to child mortality and was not controlled for by our econometric model. We, however, choose to discuss this policy in this chapter’s education section.

In conclusion, health insurance does not have a significant impact on child health in Vietnam because healthcare to children is officially free. Social protection and poverty reduction programmes cannot explain why Vietnam has low mortality for its structural factors because our earlier regression analysis has already taken income inequality (ex-post government income redistribution programmes) into account.

Education Policy

Causal Pathways between Education and Child Mortality

Education policy can only explain why Vietnam has low mortality for its structural factors if two conditions hold true. First, there must be a causal link between education and mortality which our regression analysis did not control for. And second, Vietnam must have better educational outcomes than comparable countries.

Our earlier analytical framework demonstrated that there are two causal links between education and child mortality. First, education can reduce child mortality by increasing the power of a mother relative to other members of her family. This can lead to an increase in the resources spent on children, raising their consumption on food and medical goods and hence improving nutrition and illness control. It may also increase women’s power over birth control decisions and reduce the amount of time they are prepared to spend rearing children — reducing fertility, which can in turn lead to a reduction in child mortality. Second, education can also reduce infant and child mortality by increasing women’s knowledge of, and ability to understand, health interventions, hygiene and illness control. Our econometric analysis controlled for women’s power, but it did not directly control for their level of education⁵⁸. Education policy could thus potentially explain why Vietnam has low mortality for its structural factors through this causal pathway.

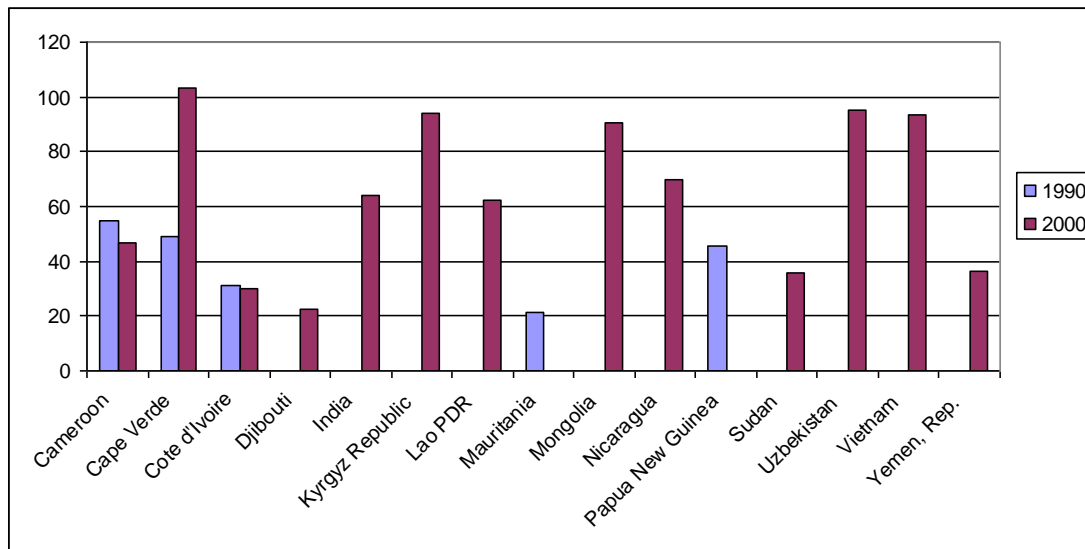
Vietnam’s Educational Outcomes in a Comparative Perspective

Vietnam also has high educational outcomes compared to similar countries. Graph 25 shows girls’ primary school enrolment rate in Vietnam as a percentage of the total number of girls

⁵⁸ Our econometric analysis did undertake some regressions which compared the explanatory power of women’s education and power, but the model through which we selected Vietnam did not directly control for women’s education.

eligible to attend secondary school in 1990 and 2000. Vietnam has a higher rate of enrollment than many comparable countries.

Graph 25: Primary Enrollment Rate, Female (% of relevant age group)⁵⁹



Education Policy and the Education System in Vietnam in the 1960s and 1970s

Female literacy and girls' primary school enrolment affect mortality through a complex lag structure. A child born in 2000 may have a 30 year-old mother who attended primary school in the mid-1970s, or a 16 year-old mother who only finished secondary school education a few years beforehand. Given that our dataset included under-five mortality data from the 1990s and 2000s, this means that educational policy from the early 1970s is directly relevant in explaining why Vietnam has low child mortality for its structural factors. The rest of this section explains the evolution of the Government of Vietnam's education policy since the 1970s to the present.

During the 1970s the government developed a socialist education system. This system was directly operated and controlled by the government and was funded by central government and local collectives. Education was provided free at the point of delivery. The government claimed to be providing universal access to education but there were some inequalities in education and not all groups received universal access (London 2006).

Research into Vietnam's education system highlights the remarkable achievements of the government's policies in making public education widely accessible and in narrowing the gender gap in education since the 1960s (Fraser 1993). By the 1970s Vietnam's indicators of basic educational achievement were comparable to countries with much higher income levels (Biddington and Biddington 1995).

The two main problems in Vietnam's education system in the 1960s and 1970s were inequalities in access to education and the low quality of provision. In most rural areas formal schooling consisted of little more than three hours on a dirt floor in a thatched hut (London 2006). And while education policies were in principle egalitarian, in actuality those with

⁵⁹ All data are taken from World Development Indicators.

political ties to the Communist Party received preferential access. There is also some evidence that villages in the south that had ties to the Republic of Vietnam regime were routinely denied access to schooling (London 2006).

Reforms to Education in Vietnam in the 1980s and 1990s

The institutional arrangements responsible for financing education in Vietnam came under considerable strain and began to disintegrate in the 1980s. The breakdown of collective economic institutions, high inflation and declining state budgets undercut the financing of education and led to a real decline in teachers' wages and declining morale in the teaching profession. There was only a minor increase in total enrolments between 1980 and 1990, even though Vietnam gained a million more school aged children during this period (London 2006).

Post 1990 a more market-oriented system of education was put in place. A fully subsidized education system was replaced by one funded partly through user fees. The cost of user fees increased dramatically throughout the 1990s; placing a considerable financial strain on households. Indeed Summerfield (1997) calculated, using VLSS data, that by 1993, just a few years after their introduction, households were covering more than 50%, 67% and 72% of the costs of primary, secondary and higher education respectively. The percentage of education costs covered by Vietnamese households was also high by international standards (Bray 1996). The cost of education also increased throughout the 1990s (Belanger and Liu 2004). For poor households the annual cost of sending a child to primary school increased from 22% to 31.5% of the non-food budget over the five years from 1992/1993 to 1997/1998 (Summerfield 1997, using VLSS data).

The introduction of user fees also caused new socioeconomic stratification in schooling⁶⁰. Before the introduction of user fees, differences in access to schooling had been correlated with wealth, but had been caused by linkages to formal sector employment and the Communist Party. Members of the Communist Party and state workers were, for example, more likely to be able to access high quality secondary schooling. The introduction of user fees caused new inequalities based on the ability to pay. For instance, by 1997/1998, the poorest households spent an average of 60% of their non-food budget for a child in lower-secondary school, while the richest households spent just 30%; meaning that upper-secondary education was not affordable for poor households (Belanger and Liu 2004, using VLSS data). Unsurprisingly secondary school enrolments are much lower for poor households and econometric regressions show that income is a significant determinant of whether or not a family sends its female children to school (Belanger and Liu 2004).

The government has attempted to mitigate the impact of user fees on enrolment through its poverty reduction programmes. The Hunger Eradication and Poverty Reduction programmes assisted poor families by providing tuition fee waivers, free books and grant scholarships so

⁶⁰ London (2006) argues that the inequalities created by the market system are a replication of earlier inequalities caused by political connections. We do not believe that this is strictly true. The current inequalities in access to education are principally caused by differences in income. Inequalities in access to education prior to the introduction of user fees were principally caused by variations in political connections and power. Certainly there is a correlation between these two kinds of inequality, mainly because in post-reform Vietnam, success in the market system (and hence income) is partly caused by access to the political elite and state. However, the new inequalities are not simply a replication of pre-reform inequalities.

that they can access upper-secondary and higher education institutions⁶¹. There is strong evidence that the tuition fee waivers lead to increased enrolments among poor households; with many poor families reporting that they would not have enrolled their children in school if that had necessitated paying fees (UNDP 2004). This programme is generally thought to be well targeted – with most people who receive support actually being poor, but limited in scope with officials only classifying 11.4 % of the population as poor because of the limited financing available (UNDP 2004). Indeed it is estimated that only one quarter of the poorest quintile and one fifth of the second poorest quintile received full tuition fee exemptions (WB 2005). The fact that local officials are only allowed to categorize a certain number of households as poor also leads to some arbitrary decisions over who qualified as poor. Poor households that are exempt from fees still face significant transport and food costs in sending their children to school.

Despite the inequalities and the financial pressures caused by user fees, their introduction did lead to substantial increases in the resources received by the education sector. Both the amount spent by households and the state on education substantially increased throughout the 1990s. These resources were used to substantially expand the number of primary and secondary schools – increasing the number of school places and improving access to education. Teachers' wages and their purchasing power also increased as did their morale (London 2006).

The increased income of the majority of households due to Vietnam's strong economic growth throughout the 1990s also meant that more families could afford to pay user fees. Although school enrolments fell in the early 1990s they increased and reached record levels from 1993. Primary enrolments also increased for all segments of the population, with more poor children attending and completing primary school (London 2006).

Despite the liberalization of Vietnam's economy and the introduction of user fees, the government still determines the overall goals and direction of the education system. For instance, in 1991, the government passed the primary education law, which made primary education compulsory for all children.

The government also developed detailed targets to ensure that provinces improved educational standards (Biddington and Biddington 1995). Provinces, communes or villages, were only allowed to claim that they had achieved universal primary education provision if they had achieved a series of enrolment and literacy targets. The government also developed a detailed curriculum to ensure that those completing primary education had achieved basic literacy and other standards. These policies have led to all but universal primary education, and in recent years the government has started to shift attention to lower-secondary education. The government is now aiming for universal lower-secondary education.

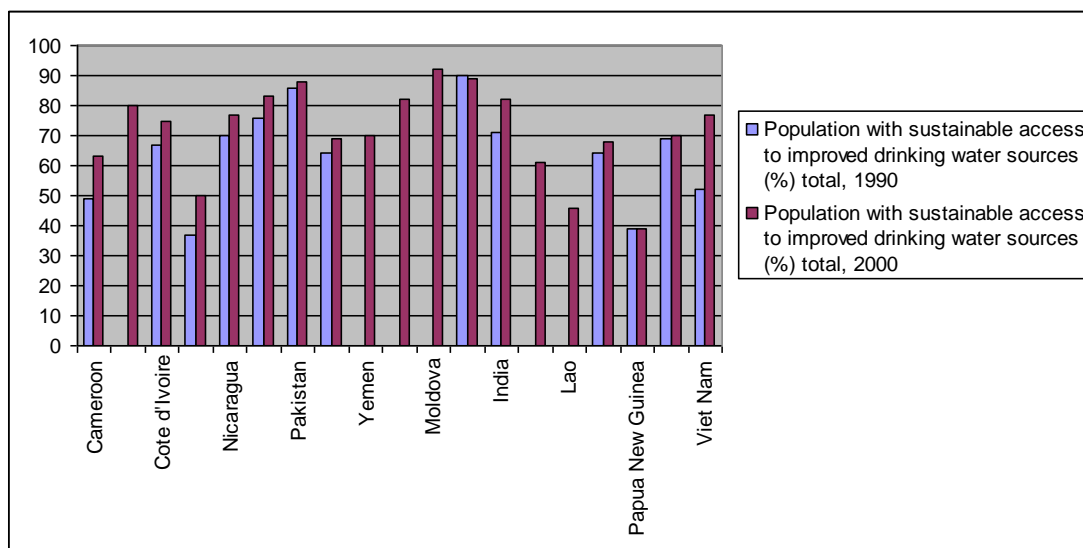
⁶¹ The government established this program in 1998 by combining Programme 133 (a previous poverty reduction programme) with the Program 120 (an employment generation scheme). In 2005 this program was renamed the National Target Program for Poverty Reduction (NTPPR) and its mandate was renewed with new goals and policies until 2010. Here we examine the goals, policies and success of the project's first phase which lasted from 1998 to 2005. We do not examine the impact of the project from 2005 onwards. There are two reasons for this. First, it is too soon to evaluate the impact of the second phase of the program. And second, the second phase of the programme post-dates the period for which our econometric analysis showed that Vietnam was an outlier. Poverty reduction policies implemented in 2006 cannot explain why Vietnam had low child mortality for its structural factors before 2005.

In conclusion, the government has long emphasized the importance of education. And it has been successful in achieving high rates of primary school enrolment and female literacy. The policies used to achieve these goals have, however, changed dramatically over time. During the 1970s the government implemented socialist policies, with education being free at the point of demand. The institutional arrangements for financing this system, and to some extent the education system itself, began to collapse in the 1980s, leading to the introduction of user fees. The introduction of user fees created new inequalities based on the ability to pay for education, but also increased funding to schools. This increase in funding improved teachers' pay and morale and the quality of education. These in turn led to an increase in demand for education stimulating enrolments. Higher enrolments and better quality education further increased literacy rates.

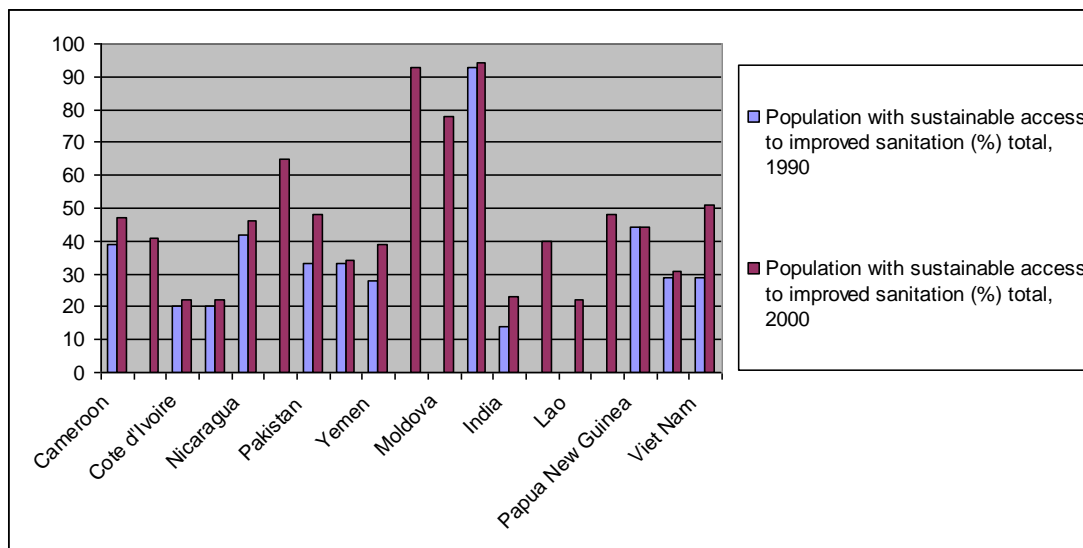
Water Supply and Sanitation

Our analytical framework showed how access to clean water supply and effective sanitation can reduce child mortality by decreasing exposure to diseases such as cholera. Water supply and sanitation cannot, however, explain why Vietnam has low mortality for its structural factors compared to other countries. The reason is that Vietnam has performed poorly in these areas. This poor performance is clearly demonstrated in graphs 26 and 27, which show the proportion of the populations with sustainable access to improved drinking water and sustainable access to improved sanitation in Vietnam and comparable countries.

Graph 26: Population with Sustainable Access to Improved Drinking Water in 1990 and 2000



Graph 27: Population with Sustainable Access to Improved Sanitation



Swinkels and Turk (2003) also support this conclusion, arguing that Vietnam's performance in the provision of water supply and sanitation has been poor compared to other countries in the region and internationally. As the aim of this section is to examine social policies that can explain why Vietnam has low mortality for its structural factors, we do not further examine water and sanitation policy here.

Conclusion

This part examined social policy in Vietnam. Our a-priori reasons for considering that Vietnam has successful social policy are that it has low (once structural factors are taken into account), and sharply falling, child mortality. The sharply declining rates of child and under-five mortality in Vietnam also mean that it is one of the few countries currently on course to achieve MDG4.

Social policies cannot directly reduce mortality. Children cannot directly die from their parents receiving a poor education. A heuristic analysis of social policy would then examine how each social policy affected mortality through a direct proximate variable. Yet for practical reasons it is impossible to undertake such an analysis. However, what we did do is create two methodological bridges between social policy and mortality. First, we defined social policy with regards to our earlier analytical framework. Second, for each social policy we outlined the theory linking it to child mortality.

We defined social policy as public policies in family planning, healthcare, water supply and sanitation and education. The main reason for defining social policy in this way was that our earlier analytical framework demonstrated that these policies can affect infant and child mortality independently of income, income inequality and women's power.

This chapter examined infectious disease and immunization policies relating to children separately from general healthcare. There were three reasons for this. First, child deaths from infectious diseases have fallen sharply in Vietnam and are now lower than in comparable countries, so social policy in this area is particularly important in explaining mortality decline. Second, because immunizations directly affect child mortality they can provide an important link between socioeconomic determinants of child mortality (such as general health policy) and proximate determinants. And third, the government established a vertical immunization programme which is managerially separate from other health policies.

Vietnam's government has a longstanding policy on immunization. Inoculations and immunizations have been provided to children free of charge through the dense network of CHC and village health workers. These policies have led to increasing, and compared to similar countries, extremely high rates of inoculations and vaccinations. These policies have contributed to Vietnam's epidemiological transition, whereby less, and a smaller proportion of children die from infectious diseases. These policies have undoubtedly led to falling, and compared to other similar countries, low mortality in Vietnam.

Vietnam's government has a long standing and continuous commitment to family planning. The ideal of a two child family norm has been promoted since the 1960s and an effective policy has been put in place since the 1980s. The three main pillars of this programme have been fiscal incentives and fines to encourage small families, the use of mass media to promote the small family ideal and the provision of free contraception through the government's health system. These policies have improved intermediate outcomes with most women knowing of, and using, contraception. Commentators have also regarded this policy as highly effective; with one study concluding that Vietnam had one of the seven most effective family planning programmes in the world (Ross and Stover 2000). Family planning policy has led to Vietnam having much lower rates of fertility than other comparable

countries and contributed to sharply falling fertility in Vietnam. Falling fertility has, in turn, lead through increased birth spacing to low child mortality. There is little doubt that the evidence presented in this chapter shows that Vietnam had effective family planning policy and that this helps explain why it has low mortality for its structural factors.

The government's family planning and immunization policies are implemented through a network of CHC, village health workers and hospitals. There is substantial evidence that this system is denser and provides better quality healthcare to children than in other comparable countries. Prior to 1986 the health system offered treatment that was free at the point of demand. Post 1986 the health system became more market-oriented with drugs mainly being supplied by the private sector, public health facilities charging user fees and the legalization of private hospitals. User fees were, however, waived for child healthcare. There is also evidence that child healthcare was cross-subsidized with health facilities using the money from user fees to increase staff remuneration and improve facilities, which in turn improved the quality of healthcare received by children.

Female education can also reduce child mortality. The government has always strongly emphasized education and prior to 1990 education was freely available at the point of demand to the majority of children. The government was successful in increasing enrolments during this period, but the standard of education was generally regarded as low, and there was some stratification in access to education based on political ties to the Communist Party. Post 1990, user fees were introduced for education. These increased funding and quality and were a substantial financial burden on households, but did not lead to a reduction in enrolments. Some poor families also received tuition fee waivers. Throughout both periods, female enrolments were higher in Vietnam than in comparable countries and most commentators regarded Vietnam as having implemented a successful education policy.

Water supply and sanitation policies and poverty reduction programmes were also examined, but these did not explain why Vietnam has low child mortality for its structural factors. That is, they are not a component of "successful social policy in Vietnam".

We also examined the distribution of social policy and child mortality in Vietnam. An equal distribution of social policy could lead to lower child mortality through the diminishing marginal returns hypothesis and thus explain why Vietnam has low child mortality for its structural factors. We might also expect that communism would have led to a more equal distribution of social policy outputs and child mortality in Vietnam compared to other developing countries.

There is, however, much evidence that egalitarian social policies cannot explain why Vietnam has low child mortality for its structural factors. Inequality in the distribution of child mortality by income increased throughout the 1990s and by 2002 Vietnam had more unequally distributed child mortality by income than other developing countries with a similar per capita GDP. This suggests that egalitarian social policy cannot explain Vietnam's low child mortality for its structural factors; in a country where all groups had equal access to social policy we would expect there to be less, and not more, variation in child mortality by income.

There is also evidence that social policy outputs are as unequally distributed in Vietnam as in other countries. More specifically, child immunizations, antenatal care and school completion

among women were as unequally distributed by income in Vietnam as in other developing countries with a similar GDP per capita by 2002.

Market-oriented reforms in healthcare, such as the introduction of user fees, could explain increased inequality in child mortality in Vietnam. They could also potentially explain why Vietnam has as unequally distributed outputs from social policies as other developing countries. The picture here is, however, complex and difficult to discern. The impact of market-oriented reforms on the distribution of child health services by income should have been limited because child health services were officially provided free of charge and were exempt from user fees. On the other hand there is evidence that many CHC charged unofficial fees which may have restricted access for poorer families. Health facilities in richer provinces may also have seen a larger increase in their revenue leading to greater inequality in the quality of child health services by income. It would also be wrong to assume that access to health services was entirely egalitarian prior to the introduction of market-oriented reforms. In the 1980s superior access to healthcare was enjoyed by government employees, urban dwellers and the politically connected (London 2006).

Overall it is clear that Vietnam does not have more egalitarian social policy, nor a distribution of child mortality that is less stratified by family expenditure, than other developing countries. So the distribution of social policy outputs cannot explain why Vietnam has low mortality for its structural factors.

In conclusion, Vietnam has effective social policies. More specifically, the chapter has provided evidence that social policies in the areas of health, education and family planning reduced infant and child mortality in Vietnam. These social policies also explain why Vietnam has low child mortality for its structural factors. That is, our econometric analysis did not produce a fluke result! A detailed analysis of specific social policies has revealed that all the evidence shows, and nearly all commentators agree, that the Government of Vietnam's social policies have effectively reduced child mortality.

5.5 State Institutions Underlying Social Policy and their Historical Development

Introduction

Vietnam has effective social policies. Our analysis could stop here. We could conclude that the reason Vietnam has low child mortality, and is on course to achieve the fourth MDG, is because of its effective social policies. Our policy recommendation to other developing countries would then be - copy Vietnam, create a dense primary health infrastructure, implement effective family planning programmes and improve female education. Adopting these policies would not guarantee that developing countries achieved the fourth MDG; for as our earlier econometric analysis showed, structural factors account for most of the decline in child mortality. But implementing such policies would minimize child mortality given countries' structural characteristics.

Yet such an answer is insufficient. For it inherently assumes that the only constraint on the ability of developing countries' governments to minimize child deaths is that they don't know what constitutes effective policies. This assumption is wrong. For as discussed earlier in this

thesis, the impact of family planning, child immunization and female education on child health has been examined in detail by numerous researchers. Donor agencies have consistently stressed the importance of these policy measures and many countries have long stated that they are committed to implementing such measures. However, despite widespread knowledge of these policies and the stated commitment of governments to implement them, there are still significant differences in the rates of child mortality between developing countries once we have controlled for structural factors.

Thus we can ask the additional question “why was the government of Vietnam able to implement social policies which reduced child mortality?” Our argument is that in Vietnam specific state institutions developed which allowed the government to control, cooperate with, and provide services to citizens in order to reduce child mortality.

A detailed analysis of all the institutions that make up the state in Vietnam would take this thesis too far from its original research question. The following paragraphs, therefore, do not describe each institution in detail but instead give brief examples of how the functioning of institutions in Vietnam led to a state that could implement effective social policies to reduce child mortality through cooperating with, controlling and providing services to, its population. It is based on a reading of secondary sources.

State Institutions and Social Policy in Vietnam

Our definition of state institutions in Vietnam encompasses the Communist Party, the government (which includes the legislative, executive and judiciary) and the mass organizations. The justification for using this broad definition is that within Vietnam the party, government and mass organizations are so closely intertwined that the way they function and operate cannot be understood in isolation from each other.

The state in Vietnam has effectively cooperated with its citizens to reduce child mortality. Through mass organizations the state has been able to mobilize large numbers of the population to assist in the implementation of its policies. Mass organizations have, for example, played an important role in Vietnam’s family planning policies which are a determinant of Vietnam’s low child mortality rate⁶². The dense structure of the Communist Party which reaches down into every town and village has also played an important role in mobilizing people to support the party’s social and economic policies⁶³.

Vietnam has also created institutions to control its population by managing political dissent and manufacturing consensus. Effectively the state controls potential opposition to its policies by ensuring that all political debate occurs within a narrow spectrum. This allows it to repress and control opposition to its policies without the need for violence. Citizens are, for example, allowed to vote in national and local elections, but all candidates are carefully vetted by the Communist Party. The end result is that all members of the House of Representatives agree with the political status quo and spend most of their time debating narrow technical questions and not directly challenging the government’s policies (Solomon 2007). This allows policies which may be unpopular with the population to be more easily

⁶² For a detailed discussion of mass organisations in Vietnam see: Fritzen (2002) Porter (1993), Sakata] (2002) and Stromseth (1998, chapters 4 & 6).

⁶³ For a more detailed discussion of the role of the Communist Party in Vietnam see Koh (2001), Kerkvliet (2005), Porter (1993), Dixon (2004) and Salomon (2007).

introduced and implemented in Vietnam than in many other developing countries (Salamon 2007 and Dang and Beresford 1998). The Vietnamese Government could, for example, implement an effective family planning policy which included fines for those who had large families even though this policy initially went against cultural norms for large families and a preference for male children. Moreover, there was little organized opposition to the implementation of this policy (London 2006 and Dang and Beresford 1998).

The dominance of policy over legality in Vietnam has also made it easier for the Communist Party to implement its policies and thus increased the state's repressive power. In the late 1980s and early 1990s policy was implemented regardless of whether or not it contradicted previous laws. In the late 1990s and early 2000s there was a movement to rule through law. Yet while this has affected the mechanisms through which Communist Party policy is implemented, it has not yet enabled citizens to challenge unpopular policies through the legal system. The judiciary is still closely linked to the Communist Party, and judges rarely make decisions which contradict its policies⁶⁴.

The structure of state institutions in Vietnam has also increased the capacity of the government to provide services. The Communist Party and state are deeply intertwined and together provide a dense institutional network which reaches down from the centre into every town and village in Vietnam. The state in Vietnam is much denser and has greater reach than that of many other developing countries. The dense network of communes, which were initially created for social and economic reasons, allowed the government to provide basic health care services which were important in reducing child mortality⁶⁵.

The legitimacy of the Communist Party in Vietnam underlies the state's power. The party's leadership during the struggle for national independence has meant that it is closely associated with nationalist sentiment and this, combined with a lack of political alternatives, means that it is regarded by many in Vietnam as the natural party of government. The party's legitimacy has also been strengthened in recent times by its success in overseeing strong economic growth and rising living standards. This legitimacy serves to minimize dissent from the population and makes it easier for the state to implement unpopular policies⁶⁶.

The underlying distribution of power between different socioeconomic groups was also conducive to the development of a powerful state. Cooperating groups of bureaucrats, the state business community and party officials dominate political power and mainly determine the direction of state policy. These individuals have a strong motivation to cooperate with each other and maintain the power of the state because their income, welfare and status are determined by their close links to the state. These groups have, for reasons of self-interest, contributed to the development of a powerful state (Gainsborough 2002).

The History of Vietnam and the Development of Effective State Institutions

Vietnam's effective state institutions, the concentration of power among a few socioeconomic groups and the legitimacy of the Communist Party are themselves outcomes

⁶⁴ For more detailed discussions of policy making and the legal system in Vietnam see Dang and Beresford (1998), Gillespie (2007), Painter (2004), and Riedel and Turley (1999).

⁶⁵ For more detailed discussions of the legitimacy of the Communist Party see Bradley (2009), Kerkvliet (2005), Dixon (2004) and Beresford (1988).

⁶⁶ This paragraph is based on Hall (1995), Coedes (1966), McAlister (1969), Beresford (1988) and Porter (1993).

of a long and complex historical process. A detailed examination of the history of Vietnam would take this thesis too far from its original research question and is therefore not undertaken. Rather, we briefly summarize key points from different periods of Vietnam's history.

The pre-colonial history of Vietnam resulted in a country dominated by a single ethnic group with a shared language and cultural heritage. Vietnam also had a long history as an independent sovereign state despite borrowing ideas and systems of governance from China and periods of direct Chinese rule. This history lends any Vietnamese government which can claim to be independent a degree of legitimacy (Kerkvliet 2005).

Colonial policies sharply increased the power of the state to repress the population. The French colonial administration implemented an effective and regressive tax system, reduced village autonomy and monitored society through a secret police force. A cadre of well educated administrators was also developed by the colonial authorities. The introduction of an effective tax system and centralized state were essential aspects of the development of a modern state which eventually underpinned the implementation of social policies⁶⁷.

The support for the Communist Party by the bureaucratic socioeconomic class meant that much of the administrative capacity within the French colonial system was not lost after the revolution. The Viet Minh and Democratic Republic of Vietnam also implemented policies which strengthened the power of this bureaucratic class; this class had a vested interest in developing a strong and effective state⁶⁸. The coming to power of the Communist Party brought with it a new philosophy with implications for state institutions, the distribution of power between groups and social policies.

Communism as an idea provided a blueprint for the development of a set of institutions to mobilize, inspire and control the masses. These institutions underpinned an effective state (Kornai 1992). Communism also provided a blueprint for policies such as land redistribution which sharply curtailed the political power of landlords and for social policies such as free universal education which directly contributed to falling child mortality (London 2006).

Communism is also an idea which implies certain social policies. It emphasizes that citizens should receive free access to basic items of consumption: in practice most communist states have had some success in providing free and universal access to healthcare and education. Social policies in the 1970s and 1980s clearly had many communist elements; with free and near universal access to child immunizations, education and family planning contributing to falling mortality (London 2006).

The importance of communist social policies in explaining Vietnam's low child mortality for its structural factors can, however, be overemphasized for four reasons. First, in our earlier regression analysis Kazakhstan and Moldova, which are former communist countries, were *negative* outliers. Second, while communism as an idea may support the universal provision of services, the actual provision of such services requires considerable state capacity. Many developing country governments which have been committed to implementing universal and effective healthcare have failed to do so (Castro-Leal et al 2003). Third, even before Doi Moi

⁶⁷ This paragraph is based on my reading of the following works McAlister (1969), Coedes (1966), Mus (1949), Beresford (1988) and Duiker (1981).

⁶⁸ This paragraph draws on work by Duiker (1981), McAlister (1969) and Turner (1975).

Vietnam's social policies deviated substantially from the universal policies promoted by communism, with government employees, urban dwellers and the politically connected receiving privileged access to services (London 2006). Fourth, more recently, Vietnam's welfare state has begun to take on liberal elements, with high-income individuals gaining privileged access to high quality services (London 2006). As shown earlier in this chapter, child mortality and many health outputs were by the early 2000s as stratified by income as in many other developing countries. Yet even as its social policies have been liberalized, Vietnam has continued to have low mortality for its structural factors.

In the Vietnam War the Democratic Republic of Vietnam had to mobilize northern Vietnam's human and productive resources to fight a foe that through American support had access to tremendous financial resources and military hardware. This required increased government control over the economy and society. Policies such as the evacuation of cities, the draft, social programmes for veterans and increased collectivization showed that northern Vietnam was already a powerful state and further increased the government's control over society and the economy⁶⁹. The Communist Party's leadership during the struggle for national independence has meant that it is closely associated with nationalist sentiment and has contributed to its legitimacy.

After victory in the Vietnam War the communist government implemented policies which radically altered the structure of society in the south by removing the economic factors around which socioeconomic groups could form. This led to the effective destruction of socioeconomic groups which may have resisted the communist government's social and economic policies. This contributed to the cohesive nature of the state and society in Vietnam in the long term and reduced future resistance to the implementation of many of the government's social policies⁷⁰. Many Vietnamese also regard the Communist party as the natural party of government due to its historic role in the struggle for national independence. Such legitimacy reduces dissent and makes it easier for the state to implement its policies.

Recent economic reforms in Vietnam have not undermined the power of the state because they enriched those socioeconomic classes of bureaucrats, party officials and business men who were closely aligned with the state. These groups had little interest in reforming or undermining the political structures of the state because they gained from the state's power to subvert the free market and create a skewed playing field. This allowed for the continuing political domination of the Communist Party and for it to continue providing services, cooperating with its citizens and controlling undesirable behaviour throughout the 1990s⁷¹.

Conclusion

In summary, Vietnam is a powerful state. The government has developed specific institutions, such as mass organizations and an intertwined party and state structure, to cooperate with, control and provide services to its citizens. The narrow concentration of power between socioeconomic groups also assists the government to implement effective

⁶⁹ This discussion of the Vietnam War is based on work by Kolko (1985), Brown (1991), Karnow (1994) and Bradley (2009).

⁷⁰ This paragraph draws on work by Beresford (1988), Bradley (2009), Thrift and Forbes (1986), Nyland (1981) and Quang (1982).

⁷¹ This paragraph is based on work by Fforde and Vylder (1996), Litvack and Rondinelli (1999), Boothroyd and Xuan Nam (2000), Tran-Nam and Pham (2005) and Masina (2009).

policies and underlies the development of effective state institutions. Through these state institutions the government has implemented effective family planning, child immunization and women's education policies; which have reduced child mortality. The effective institutions of Vietnam's state did not occur by chance but were rather the outcome of a complex historical process. Pre-colonial history, colonization, the Vietnam War, communism and economic reform all played a part in establishing a powerful state.

Chapter 6: Conclusion

The fourth MDG issued a clarion call to countries to reduce their rates of under-five mortality by two thirds between 1990 and 2015. The governments of the developing world responded to this call and committed themselves to implementing effective social policies to achieve this goal. Yet most developing countries are not currently on course to achieve MDG4. Why?

The principal answer to the question, provided by this thesis, is that developing country governments cannot implement social policies to sharply reduce child mortality because three structural factors (income, income inequality and women's power) determine most of the variation in child mortality across countries and time. In addition, our case study of Vietnam demonstrates that appropriate social programmes can play a significant additional role. Finally, we suggest, but do not argue in detail, that the efficacy of social policy is partly determined by state institutions and the distribution of power between groups. These are in turn the outcome of long, complex and unique historical processes. A state cannot in a matter of a few years become more cohesive and powerful and start implementing more effective social policies simply because its government has stated its commitment to achieving the MDGs at an international conference.

The remainder of this conclusion is divided into three sections. The first section summarizes the arguments made and evidence presented in this thesis. The second section outlines our policy conclusions. The final section discusses the implications for future research from this thesis.

Summary of Chapters 2 to 5

The second, third and fourth chapters of this thesis delineated the structural determinants of child mortality. These chapters proceeded by reviewing the existing econometric literature, constructing a detailed analytical framework and empirically testing hypotheses drawn from this framework using a panel data econometric model.

Chapter 2 reviewed multivariate models of the determinants of child mortality across countries and time. It concluded that there is substantial evidence that income, income inequality, female education/power, health outputs and water supply and sanitation determine child mortality across countries and time. There is also multivariate evidence and medical evidence that proximate variables such as birth order and spacing are significant determinants of child mortality.

There is much debate regarding the effectiveness of public spending in reducing mortality. Evidence from epidemiological, health interventions and project studies show that health expenditure can potentially cost effectively reduce child mortality. But econometric studies show that health expenditures' actual average impact across countries has been low. This thesis sought to explain why government expenditures can potentially, but on average have not, reduced child mortality.

Chapter 3 constructed an analytical framework showing the causal pathways between different socioeconomic variables and how they affect child health through proximate

determinants. Four main testable hypotheses were drawn from this analytical framework. First, income inequality, income and the power of women should explain rates of child mortality and malnutrition/growth faltering across countries and time. Second, income inequality affects mortality through the aggregation hypothesis (diminishing marginal returns). Third, it is the power of women in society and not their level of education that mainly affects mortality. And fourth, state capacity may partly determine the effectiveness of public spending in reducing mortality.

Chapter 4 tested these hypotheses using a panel data econometric model. Our time fixed effects model provided striking evidence that national income and women's power reduced, and income inequality increased, child mortality. These independent variables were significant at the 1% level and explained over 90% of the variation in mortality across time and countries.

The robustness of this model was checked using different functional forms (linear, lin-log and double log), different lag structures (zero, five or 10 years) and 10 different measures of child health/survival. Overall, 40 different regressions were estimated. National income and women's power significantly improved child health in 100% and 95% of these regressions respectively. Income inequality significantly worsened child health in 80% of them. Taken together these models provide overwhelming evidence that our original hypothesis was correct and that our results did not occur by chance or through the deliberate selection of a particular lag structure, functional form or dependent variable. National income, income inequality and women's power do explain most of the variation in child health across countries and time.

We also tested how income inequality affects mortality. In our econometric model, once the actual income of the poorest 20% and 40% of families is held constant, neither the income of the richest 20% nor income inequality significantly increases mortality. So the rich getting richer when the poor are not getting poorer does not seem to increase child mortality across countries or time. This suggests that income inequality cannot be influencing mortality through the psychosocial causal pathway or be acting as a proxy for the power of groups to influence government policy. It follows that income inequality must be associated with increased mortality due to the aggregation hypothesis.

Our model also examined the link between health expenditure and mortality. Government health spending did reduce mortality and was statistically significant, but its impact was small; with a 1% increase in government health expenditure per capita only resulting in a 0.125% reduction in mortality. Our model also demonstrated that the impact of public health spending on child health did not significantly vary with government capacity.

Yet this result should be interpreted with caution. It seems plausible that a complex interaction between politics (the process through which different socioeconomic groups use their power to shape government policy) and state capacity may determine the effectiveness of social policies. There is also much evidence to support this opinion from studies of social policy in developing and developed countries. Yet it is difficult to use econometrics to model this relationship for three reasons.

First, simple measures such as health spending per capita which are included in econometric models cannot adequately capture the full range of social policies that may affect child mortality. Second, the interaction between socioeconomic groups, government institutions

and social policy is inherently complex and difficult to measure using regression analysis. Third, countries' institutions and political systems are difficult to classify without a detailed knowledge of that country.

This thesis, therefore, proceeded with an alternative methodology. We selected a country – Vietnam – which had low mortality for its structural factors and examined its social policies, in detail.

The first part of chapter 5 discussed the data sources that are available for child mortality and its determinants in Vietnam. This included a detailed discussion of the strengths and weaknesses of data from VLSS, DHS and MICS.

Chapter 5 part 2 discussed previous studies of child mortality in Vietnam. This included reviewing studies by international organizations such as UNICEF, WB, ADB and WHO. These studies provided some evidence that poverty, women's education, water and sanitation and health outputs (e.g. vaccinations) were associated with child mortality in Vietnam. These results broadly supported our analytical framework.

Part 3 of chapter 5 discussed trends in child mortality in Vietnam. Child mortality has sharply declined and it is currently one of the few countries on track to achieve the fourth MDG. This makes it an interesting country for a case study analysis.

The distribution of child mortality by income, region and ethnicity was also discussed in part 3. Child mortality is as stratified by income in Vietnam as in other developing countries with a similar GDP per capita. Health outputs such as child vaccinations are also as stratified by income as they are in other developing countries. This suggests that egalitarian social policies cannot, through diminishing marginal returns, explain why Vietnam has low child mortality for its structural factors.

There is significant variation in child mortality by region in Vietnam. Our analysis demonstrates that poverty and access to vaccinations are the most likely determinants of child mortality across regions. Ethnic minorities have a higher rate of child mortality than the ethnic majority population. We provide evidence that this is because ethnic minority groups are poorer, have lower levels of education, worse access to clean water and sanitation, higher fertility and worse access to high quality health services than the majority of the population.

The fourth part of chapter 5 examined social policies. It argued that the government in Vietnam had implemented effective social policies in the areas of family planning, child immunization and female education.

The core elements of Vietnam's family planning programme were fines and fiscal incentives that changed the number of children that families rationally decided to have, mass media campaigns which effectively promoted the idea of a two child family and the free provision of contraceptives. Such policies were effective with fertility sharply falling and Vietnam having lower fertility than most other countries with a similar GDP per capita by 2000.

The government also increased rates of child immunization throughout the 1980s, 1990s and early 2000s. Inoculations and immunizations were provided free of charge through the public health system. The government has also implemented a longstanding malaria control programme. Throughout the 1990s and early 2000s the cornerstones of this programme were

the free provision of drugs and sprayed mosquito nets, and an education programme aimed at village heads and village health workers.

Prior to 1986 Vietnam's health sector was publically funded and healthcare was available free of charge at the point of demand. Broad-based and fairly equitable access to primary healthcare, child immunization and family planning were available through a dense network of CHC. This undoubtedly contributed to falling child mortality.

During the 1980s the government liberalized the health system. Child health services were, however, not subjected to the full vigour of the market. CHC were saved from collapse by the government's decision to directly fund some of their expenditure from general revenue. Vertical health programmes such as family planning and child immunization continued to be officially supplied free of charge. The increase in funding hospitals and CHC received, from the user fees they charged for other services, meanwhile, led to increased spending on health infrastructure and remuneration for health professionals. So the reform process effectively led to payments for other health services subsidising the family planning and child health programmes.

The government has, since the 1960s, emphasized female education. During the 1970s the government developed a system of schools providing free education at the point of demand; this contributed to Vietnam having higher rates of female enrolment and literacy than many countries with a similar level of income. The standard of education was, however, low and there was stratification in access to education based on political ties to the Communist Party. The institutional arrangements for financing education and to some extent the system itself began to collapse in the 1980s. In response, the government introduced user fees. The introduction of user fees created new inequalities based on the ability to pay for education, but it also increased funding for schools, teachers' pay and the quality of education. This in turn increased demand and enrolments. Some poor families were also granted waivers from tuition fees. Female enrolment and literacy continued to increase throughout the 1990s and early 2000s and Vietnam's rates of female literacy and enrolment were in the mid-2000s still higher than in other countries with a similar GDP.

Vietnam has had effective social policies. There is considerable evidence that public policies in the areas of health, education and family planning reduced child mortality. These social policies also explain why Vietnam has low child mortality for its structural factors. This supports our earlier argument that social policies would explain why Vietnam had low child mortality for its structural factors in our panel data econometric analysis.

The last part of chapter 5 summarized the state institutions which underlay the formulation and implementation of social policies in Vietnam. We suggested that carefully controlled elections, the dominance of policy over legality, the domination of the Communist Party over the executive and legislative, mass organizations and a narrow distribution of power between groups were all important in explaining the implementation of social policies. These were the outcome of a long and complex historical process. Pre-colonial history, colonization, the Vietnam War, communism and economic reform all played a part in establishing a powerful state.

Implications for Policy

This thesis investigated the structural determinants of child mortality and analyzed social policy in Vietnam. This section discusses the implications our research has for policies governments and international organizations should pursue to achieve MDG4.

Our research has implications for policies in two areas. The first is the social policies that governments should implement to achieve MDG4. The second is the reforms that should be implemented to develop the type of states that can effectively implement these policies. In both areas, we contrast our policy conclusions with those that the international development community recommends, and to which the governments of many developing countries have a stated commitment to implementing.

The Global Strategy for Women's and Children's Health 2010 (GSWCH) outlines a strategy for achieving MDG4. Many developing countries and international donor agencies have committed themselves to following the policies outlined in this document. And, as such, this strategy can be regarded as describing a broadly accepted consensus on the best way to reduce child mortality.

The GSWCH outlines actions in six broad areas. These are support for a comprehensive integrated package of health services; innovative and increased financing; strengthening health systems and building capacity of health workers; country led health plans; improved monitoring and evaluation; and promotion of human rights, gender equality and poverty reduction.

The GSWCH recognizes that gender equality and poverty reduction can reduce mortality. But these policies are ascribed subsidiary importance to health interventions and policies. The document stresses that health policies and interventions can drive large reductions in child mortality and assist countries to achieve MDG4. Our econometric evidence does not support this emphasis on healthcare. Income, income inequality and women's power drive changes in mortality across countries and time. This conclusion is supported by other statistical studies that emphasize the importance of these structural factors in explaining child mortality across countries, time and families. Our econometric models also showed that these structural variables mainly affected mortality through their impact on family income and its distribution between family members. The importance of structural factors and the fact that they do not principally affect mortality through health policy implies that the health strategies outlined in the GSWCH are unlikely to drive large changes in child mortality.

The GSWCH calls for significantly more money to be spent on child health. This policy can also be questioned because our econometric results demonstrated that health spending has a significant, but extremely small, impact on child mortality across time and countries. This result is also supported by earlier econometric work and by a range of studies that demonstrate that health spending, even in countries committed to improving child health, often benefits the rich and powerful and may be concentrated on expensive tertiary healthcare.

The GSWCH also stresses the provision of a basic package of healthcare. It argues that this package should include family planning; antenatal, postnatal and child care; the

prevention of HIV/AIDS; vaccination and immunization; treatment of major infectious childhood diseases and treatment of diarrhoea and under-nutrition in children. Our case study provides some evidence to support these policies. The Government of Vietnam's policies in the areas of family planning, child immunization and control of infectious diseases were all important in reducing child mortality in Vietnam and explaining why it has low mortality for its structural factors.

The family planning policies recommended by the GSWCH are, however, somewhat different from those implemented in Vietnam. Family planning in Vietnam provided broad-based access to free contraceptives, but it also changed the number of children families wished to have through fines and fiscal incentives and mass media campaigns. The government deliberately and successfully sought to change society's attitudes towards small families and elements of its programme were repressive. In contrast, the GSWCH places heavy emphasis on the provision of contraceptives to meet an unmet demand for family planning. It does not explicitly recognize that mass media campaigns that change societal attitudes and fines and fiscal incentives that alter the number of children families rationally want are important elements of successful family planning campaigns. Without these elements the family planning policies promoted by the GSWCH are unlikely to curtail fertility as sharply, or reduce child mortality as significantly, as those policies we examined in Vietnam.

The importance of international donor agencies and UN bodies (e.g. the Global Fund) is also stressed by the GSWCH. Our case study of Vietnam offers little support for international organizations playing a major role in improving child health. The family planning, female education and child immunization policies in Vietnam that contributed to sharply falling rates of mortality were driven by the government and implemented through its dense primary health network. Vietnam made substantial progress in these areas prior to the 1990s when it was to a large extent isolated from the international donor community. One lesson from Vietnam is that it is important for the government to lead in the formulation and implementation of social policies.

The GSWCH does also emphasize country-led health plans and accountability. Yet here there is some tension with GSWCH also recommending the policies countries should implement. A document that outlines the policies national governments should follow cannot also be promoting national accountability; unless it is assumed that prior to the formulation of the GSWCH every country in the world had a national policy debate and that all these debates came to the same conclusion regarding the health policies that should be implemented. This would seem unlikely. Did the populations of Vietnam, Nigeria and Timor-Leste really engage in policy debates concerning the role of family planning in reducing child mortality and come to the same conclusion? Or is it perhaps the case that international conferences, the overall aim of which is to get all governments to agree to pursue the same goals in the same way, undermine national accountability? And is it not also the case that not all countries can have country-led health plans and yet also be committed to following a one-size-fits-all global strategy? We would argue that the GSWCH and MDGs, far from promoting national accountability, represent an attempt to get the governments of developing countries to sign up to a one-size-fits-all set of

policies and goals regardless of the views of their own population or their capacity to implement such policies.

The importance of civil society is stressed by the GSWCH. Vietnam is an interesting case in this regard. Social policies were implemented through the government's public health system and independent civil society organizations did not play an important role in reducing child mortality. Mass organizations, however, did play an important role in promoting the government's social policies, but they were by no means independent from the government. Rather they are, both in terms of personnel and funding, closely linked to the Communist Party and executive. Nor are mass organizations the type of organizations the international donor community envisages when it stresses civil society's role in improving child health. Our case study offers scant evidence that independent civil society organizations are essential for sharply reducing child mortality.

This thesis has also suggested that effective social policies are fostered by powerful or high capacity states. The GSWCH, apart from an emphasis on accountability, has relatively little to say concerning the types of state that may best be able to implement effective social policies. The Good Governance Agenda, promoted by many donor organizations, represents a mainstream and widely accepted view concerning the types of state that are desirable and best able to implement effective social policies. It is, therefore, instructive to compare the different policy implications of the Good Governance Agenda and our description of a powerful and cohesive state.

There are different interpretations and descriptions of good governance. Most publications by international agencies emphasize the following aspects of good governance⁷². First, the political system should be accountable and democratic with a broad-based consensus and the views of the poor underlying the implementation of policies. Second, that there should be respect for the rule of law by the government and society and little corruption. Third, that social policies implemented by the government should provide equitable and universal social services and/or be pro-poor⁷³.

Our case study of Vietnam offers little support for the idea that the views of the poor are important for the formulation or implementation of effective social policies. The power to influence policy in Vietnam was in past years, when many effective social policies were implemented, concentrated among a narrow group of bureaucrats and state officials. This group was by no means the poorest in society. Since the onset of the economic reforms, cooperating groups of bureaucrats, Communist Party officials and the state-business elite have dominated policy making and political power. The poor have not decided Vietnam's economic or social policies.

⁷² These definitions are drawn from UNDP (1997) page 12 and DFID (2001) page 9 and to a lesser extent USAID (2005). See also UNDP (2006) and DFID (2007). Grindle (2007) provides a useful summary of the different definitions of good governance.

⁷³ Although not explicitly stated in much of the good governance literature there appears to be the assumptions that countries will have a market economy. There is also an emphasis on decentralization among some donors (USAID 2005).

Our case study also appears to offer little support for the role of accountability in improving health. The government of Vietnam is not democratic and nor is it especially accountable to its citizens. Elections are held by candidates who are carefully vetted by the Fatherland Front and represent an artificially narrow range of views. Policies in Vietnam are formulated by the closely integrated Communist Party and executive with limited open debate. Vietnam's Communist Party as a mass party does include many workers and peasants as members, but there is little evidence that these socioeconomic groups have driven policy. The legislative, meanwhile, is dominated by the Communist Party and spends most of the time debating narrowly defined technical issues and not holding the executive to account. For the most part the social policies of the Party are not seriously questioned.

The importance of legitimacy is, however, emphasized in our case study. The Government of Vietnam may not be particularly accountable to its citizens, but they do regard it as legitimate. The Communist Party, through its leading role in establishing independence, long term rule, propaganda and control of the media and recent successful economic policies, is regarded as legitimate by much of the population.

The idea that respect for the rule of law by the government underlies effective social policies may also be questioned. In Vietnam during the 1970s, 1980s and early 1990s the party and executive dominated the legal system. Law was not regarded as being above the executive or Communist Party but rather emanating from it. Policies which contradicted previous laws were implemented often without challenge and so few laws were passed by the national assembly that even a semblance of reality ceased to exist. Effective social policies were formulated and implemented in a country where citizens had to obey the state, but where the Communist Party and executive could ignore the law.

The court system also provides citizens with limited opportunity to hold the government to account. Court officials are vetted by the Communist Party and most are also party members. The courts are officially subordinate to the National Assembly which is dominated by party members. In the 1980s and early 1990s legality did not constrain the Communist Party's or the executive's ability to implement policy. Communist Party members and state officials are, moreover, rarely disciplined through the court system for corrupt or other illegal activities. The system of governance is one whereby the Party-State can formulate and implement effective policies but it is not particularly accountable to its citizens.

Our case study also offers mixed support for the argument that effective social policies have to be popular. The government's programmes and policies to implement immunizations and malaria treatment were broadly popular and encountered little resistance from society. Yet its family planning policies went against deeply held views within society. Traditional Vietnamese culture, prior to the implementation of family planning programmes, emphasized the importance of large families and a preference for sons. It was only through the consistent promotion of the ideal of the two child family through the mass media, coupled with fines and fiscal incentives to encourage small families, that the government was able to change these views. The government even went as far as sacking civil servants who had more than two children. There is little evidence that such family planning policies were popular or dependent on a broad-based consensus. Rather they involved the Communist Party going

against the wishes of society to implement unpopular and controlling policies. Yet family planning policies contributed to sharply falling fertility and lower child mortality.

It is difficult to draw any simple lessons from our case study concerning whether social policies have to be universal or pro-poor to drive changes in mortality. The policies implemented by the Communist Party in the 1970s, 1980s and early 1990 did offer near universal access to immunizations and basic healthcare through the government's dense network of commune health workers. Communism as an ideology, which can be regarded as supporting universal access to basic services, was an important element supporting such policies. There was, however, always a degree of stratification in access to health and education based on political power and ties to the party state. In recent years new barriers to access based on ability to pay have occurred; with richer sections of society paying for superior care. Child mortality rates and children's access to immunizations are now as stratified by family income in Vietnam as they are in many other developing countries.

Therefore we conclude that our case study does not support the Good Governance Agenda⁷⁴. It was not good governance, but a powerful state, which underlay effective social policies in Vietnam. These were implemented by an undemocratic state, the Communist Party and mass organizations. The Communist Party's dominance over the executive, legislative and mass organizations has underpinned Vietnam's social policies. Such institutional arrangements do not conform to the good governance model proposed by the international development community.

State power and good governance are not equivalent. It is possible for states such as Vietnam, China and South Korea (in the 1970s) to be powerful in the sense that they can provide services to, control and cooperate with, their populations but also embody many elements of "bad governance" such as low accountability, limited democracy and a government which is above the law. It is, moreover, a narrow distribution of power between a few cooperating socioeconomic groups which underpins such states. These are not states which provide opportunities for all poor people to influence policy. Good governance and powerful cohesive states are not different labels for the same or similar things. And there is evidence that it is state power and not good governance that explains the effectiveness of social policy.

Does the difference between powerful and good governance states matter from a policy perspective? The answer is only if the policies that countries and international organizations follow in pursuit of good governance differ from those that lead to the development of powerful cohesive states.

At the heart of good governance policy is the idea that donors should recommend, and developing governments should implement, certain reforms that lead to better and good governance. Donors may attempt to analyze the recipient's political situation and history⁷⁵, discuss options with its government and review state capacity before formulating a

⁷⁴ This conclusion is supported by Painter (2004) who argues that Vietnam's stellar economic performance and poverty reduction have occurred despite its government being authoritarian and scoring poorly on good governance indicators. He further argues that good governance reform is not, and perhaps should not, be a top priority for the Government of Vietnam.

⁷⁵ See, for example, DFID's drivers of change analyzes.

recommendation⁷⁶, but the output is always a list of reforms agreed with the government⁷⁷. Technical assistance projects then will be devised to help achieve this goal⁷⁸.

There is little evidence⁷⁹ from Vietnam that deliberate attempts to reform the state, sponsored by technical assistance, were crucial or key to the development of state power⁸⁰. State institutions and power in Vietnam were the outcome of a long term historical process. Historical events as tumultuous as colonization, communist revolution and the Vietnam War were the driving force behind the development of a powerful state with high legitimacy and a narrow distribution of power within society. Communism as an external idea fermenting revolution, encouraging a redistribution of power, embodying certain social policies and providing a model for state institutions was an important factor. But donor sponsored ideas and technical assistance programmes encouraging good governance played, compared to the grand events of history, almost no role. There is scant evidence from Vietnam that the donor agenda for good governance will by accident lead to the development of a powerful state.

The recognition that history moulds countries does not mean weak states will persist forever. Recognising that historical events, and not technical assistance or pledges given at international conferences, determine state power and the efficacy of social policy is not equivalent to path dependency. Vietnam is a cohesive state which might become a democratic multi-class state. The historical process through which such transformations in state types may occur is complex and we do not attempt to predict the future here. But what we can confidently say is that such state transformation will not occur due to the policies of donor organization.

The policy conclusions from our thesis can be summarized as follows. First, structural factors matter. Income, income inequality and women's power explain most of the differences in mortality across countries and time. Social policies' impact on mortality is thus limited. Second, social policies in the areas of family planning, female education and child immunizations can reduce mortality. Third, power matters as much as policy. A powerful cohesive state must underlie the implementation of effective social policies. Such states may have elements of bad governance but they can cooperate with, provide services to, and control, their populations. Fourth, such powerful cohesive states are created by tumultuous historical events and not donor-sponsored reforms and technical assistance.

⁷⁶ Donor documents often claim that they take account of local capacity. WB (2004) page 194 advises "choosing and sequencing public sector reforms carefully, in line with initial capacities, to create firmer ground for further reform". However, whether international donors actually follow this advice is a matter of some debate. Acemoglu (2008) argues that while there are some indications that the WB tailors its approach to specific national conditions, broad-based non-country-specific interventions still remain the norm.

⁷⁷ Fukuyama (2007) criticizes the donor driven reform effort arguing that while principles of good governance are laudable, in practice reforms are essentially donor driven and technocratic. Reforms, moreover, are developed in such a way that they are often neither appropriate nor realistic.

⁷⁸ There is also much debate regarding the extent to which good governance can actually be measured. Chibba (2009) argues that the reliability of measurements of good governance is poor in many countries.

⁷⁹ It could also be argued that there is little evidence that international donors are substantially improving governance. The WB (2006) notes "in spite of improvements [in governance] in a number of countries there have been a similar number of countries where a deterioration has occurred".

⁸⁰ Similarly Goldsmith (2007) challenges the validity of governance reform as a catalyst for development. He argues that international development agencies have underestimated the time and political effort required to change governance and overestimated its impact on economic growth. Ayeni (2000) argues that good governance cannot be imported and is instead the production of the local environment and its history.

These policy conclusions imply that the international development community should recognize the limitations developing countries face in using social policies to achieve MDG4. The importance of structural factors means social policies' scope for reducing child mortality is limited. And the effectiveness of social policy is determined by state power and the broad sweep of history. Developing countries cannot implement social policies to suddenly increase the rate of reduction in under-five mortality. And given that most countries are not currently on course to achieve MDG4, this means that they cannot achieve this goal. It is already too late. Some might argue that there is nothing wrong with ambitious targets, but there is an inherent danger in MDG4 that we are holding many governments to a target that they cannot possibly keep. And that this may lead in 2015 to one of the development community's periodic bouts of self-induced depression where it bemoans the failures of development and wonders if all that Official Development Assistance was worth it. Given the importance of history and structural factors, the reader may wish to carefully consider whether getting the developing world's leaders to solemnly pledge to reduce child mortality by two thirds was a meaningful endeavour. My conclusion is that MDG4 was of no use at all.

Implications for Future Research

This thesis has argued that countries' social policies explain whether they have high or low mortality for their structural factors. This argument was supported by a case study of Vietnam. Concentrating on a single country allowed us to undertake a detailed and nuanced analysis of its social policies. It is, nevertheless, the case that while social policies do explain Vietnam's outlying status we cannot regard a single case as providing overwhelming evidence that these variables also explain other countries' low or high mortality. Future research could, thus, extend this analysis to other countries. Do social policies explain why other countries were outliers in our regression analysis? Do social policies, for instance, explain why Moldova has low mortality for its structural factors and why Bangladesh and Kazakhstan have high mortality for their structural factors?

Future research could answer these questions using the methodology outlined in this thesis. More specifically, a detailed analysis of family planning, child immunization and women's education in Moldova and Bangladesh could be undertaken. The extension of the methodology employed by this thesis to an analysis of social policies in other countries would also allow us to make comparative observations. The effectiveness of social policies between different countries could be compared.

The methodology employed by this thesis also has implications for future research. A key component of our methodology was using econometrics to control for structural variables and then analyzing outlying cases in more detail using the case study methodology. This allows econometrics to be used for what it is good at; namely identifying simple, deterministic relationships for which accurate data exist for many units. And it also allows us to use the case study methodology for what it is good at; namely investigating complex and nuanced interrelationships where accurate statistical data do not exist and concepts are hard to measure.

Combining econometrics and the case study methodology also reduces the danger that the case study will falsely affirm the researcher's preconceived notions. Randomly selecting a case (e.g. Vietnam) and then arguing any change in the dependent variable the researcher is

interested in (e.g. mortality) was due to a selected independent variable (e.g. social policy) runs the risk of falsely ascribing changes in the dependent variable to the independent variable chosen by the author, when such changes may actually have been caused by other unobserved factors. Our methodology, by controlling for all likely significant independent variables, reduces the likelihood that a case study will erroneously confirm the researcher's pre-conceived notions.

There are significant advantages to using econometrics to identify outliers for case study research. This methodology is, in our opinion, greatly underutilized in the social sciences. Such a methodology is particularly useful when the question being asked is partly explained by easily measureable statistical variables, which lend themselves to econometric analysis, and partly by complex policy variables. A question such as *what explains rates of absolute poverty across countries?* is, for example, likely to be partly answered by a country's GDP (which can be captured in a cross-country regression analysis), and partly determined by the implementation of welfare policies. Such policies are in our opinion too complex to be adequately captured through a regression analysis. A mixed methodological approach is a powerful tool for answering such questions.

Analysis of a complex problem using econometrics and a case study is a substantial undertaking. It requires that the researcher has a detailed understanding of econometrics and the case study methodology. The time and space that can be devoted to the econometric analysis is likely to be less than in a study which concentrates on solely using the econometric methodology. Likewise, the space and time that can be devoted to the case study analysis is likely to be less than in a study which concentrates solely on using this methodology. We would, however, argue that the benefits of combining econometric and case study analysis more than outweigh these costs.

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Appendices

Appendix 1: Technical Note

The aim of the first regression is to identify countries where government health policy has been effective and ones where it has been ineffective. This method of selection ensures that we don't erroneously subscribe low infant mortality to effective government policy when in reality it is due to high income per capita or an egalitarian distribution of income. There are two methodological problems with this approach.

First, in effect we are deliberately omitting a theoretically sound variable - health policy. We are then taking the unexplained variation in the regression as a measure of health policy and examining this through a political economy analysis. Of course if health policy is correlated with the explanatory variables, then they will be absorbing some of its explanatory power (a classic case of omitted variable bias). This means that the regression may systematically understate the impact of government health policy on infant mortality.

Second, we are attributing the unexplained variation in the regression analysis to health policy. But this variation could be explained by other variables that haven't been included in the regression analysis, leading to an overestimation of health policies' impact on infant mortality.

These biases are intrinsic to our methodology and cannot be easily ameliorated. The only alternatives to using this methodology are to use an inaccurate proxy of health policy in the regression - which for the reasons explained earlier is unacceptable - or to conduct the political economy analysis without first delineating the extent that health policy determines infant mortality - which is also unacceptable.

It should also be noted that this regression is not theoretically different from explaining any dependent variable (Y) by any vector of independent variables (X), when it is known that X doesn't contain every variable that could theoretically affect Y. For example, many academic papers plot per capita GDP and infant mortality on a graph. They then regard deviations from the graph's line of best fit as an indicator of social policies' effectiveness. This methodology suffers from both biases described above, as it excludes variables that could theoretically affect infant mortality and prescribes their impact to social policy.

Appendix 2: Summary Table for Literature Review

Paper	Data Source for IMR and CMR ⁸¹	Sample Size	R - Squared (adjusted if available)	Dependent Variable	Independent Variable 1	Independent Variable 2	Independent Variable 3	Other Independent Variables
Rodgers 1979	UN Demographic Yearbook	56	0.758 (CMR) and 0.581 (IMR)	IMR and CMR	1/ GDP per Capita	Gini Coefficient	1 / GDP per Capita Squared	
Flegg 1982	UN Demographic Yearbook	46	0.697	IMR and CMR	Coefficient of the Variation in Incomes	% of Women aged 15 or over who are Illiterate	Nurses per 1000 of the Population	Physicians per 1000 of the Population
Hobcraft et al 1985	World Fertility Surveys	39 Separate Countries	N/A	NMR, PNMR, IMR ⁸² , CMR	Mother's Education	Teenage Mother	Birth Spacing	Birth Order
Waldmann1992	UN Population and Vital Statistics Report	57	0.79	IMR	1970 Indicator Variable	Log Poor Income	Log Middle Income	Rich Share
Singh1994	World Development Reports	25	0.89	CMR	% of Births Attended by Trained Personnel	Women's Labour Force Participation	Women's School Enrolment	GNP per Capita

⁸¹ Infant Mortality Rate (IMR) and Child Mortality Rate (CMR)

⁸² Neonatal Mortality Rate (NMR) and Perinatal mortality rate (PNMR)

Paper	Data Source for IMR and CMR ⁸¹	Sample Size	R - Squared (adjusted if available)	Dependent Variable	Independent Variable 1	Independent Variable 2	Independent Variable 3	Other Independent Variables
Pritchett and Summers 1996	World Development Report data classified as reliable by King and Rosenzweig (1991)	248	0.795	IMR and CMR	GDP per Capita	Years of Schooling		
Shen and Williamson 1999	UNICEF 1992 and WDI 1997	60	60	CMR	Child Survival 1960	Energy Consumption per Capita	Population per Doctor	Economic Growth, State Strength, Secondary School Enrolment, Female Secondary School Enrolment, Maternal Mortality and Contraceptive Prevalence
Filmer and Pritchett 1999	UNICEF 1992	104	0.954	IMR and CMR	Log GDP per Capita	Log of Share of Public Spending as a % of GDP	Level of Female Education	Whether a Country is Muslim and Ethnic Linguistic Fractionalization
Desai and Alva 1998	DHS	22 countries	N/A	IMR	Husband's Education	Access to Piped Water	Toilet Attenuate	
Easterly 1999	World Resource Institute (1994)	444 (IMR) & 355 (U - 5)	0.78 (CMR) 0.58 (IMR)	IMR and CMR	GDP per Capita			

Paper	Data Source for IMR and CMR ⁸¹	Sample Size	R - Squared (adjusted if available)	Dependent Variable	Independent Variable 1	Independent Variable 2	Independent Variable 3	Other Independent Variables
Rutstein 2000	DHS	89 Surveys, 56 Countries	0.684 (IMR) 0.614 (CMR)	IMR and CMR	Fourth Birth Order or Higher	Age of Mother at Birth 35 or Above	Birth Interval less than 24 Months	Medical Birth Attendance, Measles Vaccination, Medical Facility for Diarrhoea, Medical Facility for Acute Respiratory Infection, Medical Facility for Fever, Median Duration for Breastfeeding under 4 Months, Breastfeeding and Solids 7 - 9 Months, Stunted, Wasted, Dirt Floor, Primary Education and Electricity Connection
Younger et al 2001	DHS and Global Development Network	54	Only gives for period	IMR	Beginning of Period IMR	log (Real GDP per Capita)	Primary School Enrolment Rate	DPT Vaccination
Wang 2002	DHS	41	0.88	CMR	Log GDP per Capita	Five Year moving Average of Rural Population	Health Expenditure as % of GDP (from WDI)	Know about Oral Rehydration Therapy, Proportion of Children Vaccinated in the First Year of Life, Proportion of Children with Access to a Flush Toilet at the National Level, Electricity Total, Proportion of Households Linked to Electricity at the National Level,
Rajkumar and Swaroop 2007	World Development Indicators	228	0.95	CMR (under-five)	Log GDP per Capita	Log Public Expenditure Interacted with Corruption	Female Education	Income Inequality, Female Education, Muslim, Ethnic Linguistic Fractionalization, Access to Safe Water, Degree of Urbanization, % of Population Under 5, Distance from Equator,

Paper	Data Source for IMR and CMR ⁸¹	Sample Size	R - Squared (adjusted if available)	Dependent Variable	Independent Variable 1	Independent Variable 2	Independent Variable 3	Other Independent Variables
								Time Dummies,
Fay et al 2003	DHS	175 (43 countries in 4 asset quintiles)	IMR (0.82) CMR (0.86)	IMR and CMR	GDP per Capita	Ethno - linguistic Fractionalization	Gini Index	Female Illiteracy Rate, Underweight, Low Mother BMI, Access to Piped Water, Asset Quintiles 1 to 4
Hanmer, Lensink and White 2003	WDI	79 to 56	N/A	IMR and CMR	Log per Capita Gross National Product	Literacy Female to Male	Tuberculosis Vaccination	Physicians per Thousand People
Asafu-Adjaye 2004	WDI	46 countries 6 periods 264	0.989	IMR	Income lagged by one period	Gini Coefficient lagged by one period	Savings lagged by one period	Education lagged by one period
Shandra et al 2005	WDI	59	0.9	CMR	Child Mortality in 1980	log of GDP per Capita in (1980)	Educational Attainment (Gross Secondary School Enrolment for (1980)	Level of Multinational Corporate Penetration (1975)
Heaton et al 2004	DHS	42 countries giving 160,000 observations	0.073	IMR	Size of Family	Socioeconomic Index	Mother's Education	Communication, Health Utilization, Fertility 1st Birth, Birth Interval, Breastfed

Paper	Data Source for IMR and CMR ⁸¹	Sample Size	R - Squared (adjusted if available)	Dependent Variable	Independent Variable 1	Independent Variable 2	Independent Variable 3	Other Independent Variables
Anand and Baernighausen 2004	WHO, WDI (1997) , HDR (2003)	117	IMR (0.812) UMR (0.814)	IMR and CMR	Gross National Income per Person	Human Resources for Health Density		
Ram rerun of Rodgers 2006	WDI (2002)	108	0.81	IMR	1/PCY	1/PCY^2	Gini Coefficient	
Ram rerun of Flegg 2006	WDI (2002)	74	0.84	IMR	ln (Y)	Ln (Gini) Adjusted Gini Coefficient	ln (Female Literacy)	
Ram rerun Waldmann 2006	WDI (2002)	108	0.88	IMR	ln(PCY)	(Positive) Income or Consumption Share of Top 10%	DCON (Positive)	
Ram rerun of Mellor and Millor 2006	WDI (2002)	66	0.66	IMR	Secondary School Enrolment Rate			
Ram rerun of Mellor and Millor, with a different functional form 2006	WDI (2002)	66	0.91	IMR	log(Gini)	ln(PCY)	Ln (Secondary School Enrolment Rate)	
Jamison et al 2006	WDI (2002)	47	0.476	IMR	Time	Education	Logged Income per Capita	Tropical and Equest (Technological Progress Education + Trade Openness)
Franz and Fitzroy 2006	WDI	61	0.84	CMR	Calories Available per Day	Mean Consumption of the Poor	Female Participation Rate (Female Share of the Labour Force)	Female Literacy, Incidence of Tuberculosis, Estimated Total fertility Rate, Regional Dummy Variables

Paper	Data Source for IMR and CMR ⁸¹	Sample Size	R - Squared (adjusted if available)	Dependent Variable	Independent Variable 1	Independent Variable 2	Independent Variable 3	Other Independent Variables
McGuire 2006	United Nations Project, UNDP Human Development Report and WDI	46	0.723	CMR (under-five)	Log GDP per Capita	Female Schooling	Income Inequality	Ethnic Linguistic Fractionalization
Lazarova 2006	US Census International Database	112	0.72	IMR	GDP	1/GDP Squared	Governance	
Schell et al 2007	WDI	105	0.91	IMR	GNI	Young Female Illiteracy	Gini Index	Poverty Rate (not significant) and Public Spending on Health (not significant)

Appendix 3: Statistical Comparison of Different Child Mortality Datasets

This analysis compares WDI and DHS data. The box plots in graph 2 show that the two datasets have similar means and variances for every decade from the 1960s⁸³. The significant difference between the datasets in the 1960s could be explained by one of two factors. First, it is reasonable to assume the most developing countries faced very severe capacity constraints in recording births and deaths in the 1960s and therefore WDI may be more inaccurate in this, than later decades. Second, DHS data from this decade are, in most cases, lagged by more than 10 years from later surveys, as discussed earlier, this may lead to the data underestimating infant and child mortality. There are theoretical reasons for believing that DHS data are more accurate than WDI data, but our empirical results show that in actuality there is little difference between them⁸⁴.

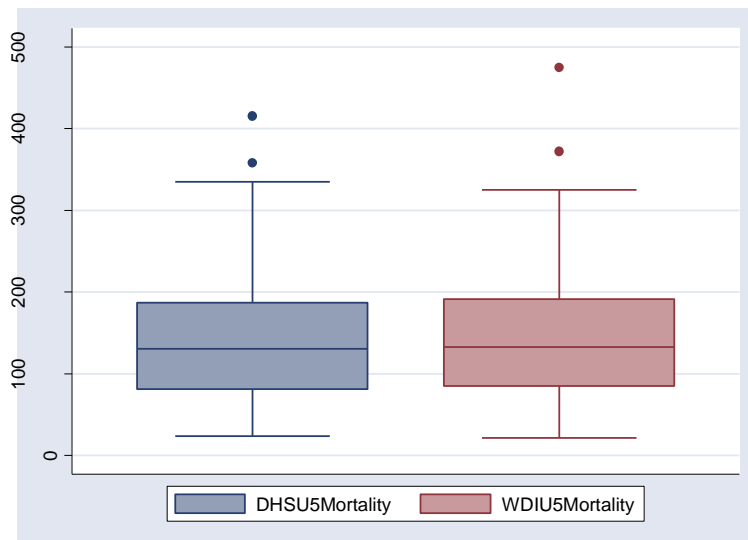
⁸³ Only countries and years which contained data from both DHS and WDI were included in this analysis.

⁸⁴ It follows that regression analyses that use either dataset for the same sample of countries are liable, unless they rely heavily on data from the 1960s, to produce similar results. The results reported in the rest of this thesis using DHS and WDI data are not, however, the same because they are for different samples of countries. We use WDI data for all countries for which they are available. And DHS data for all countries for which they are available.

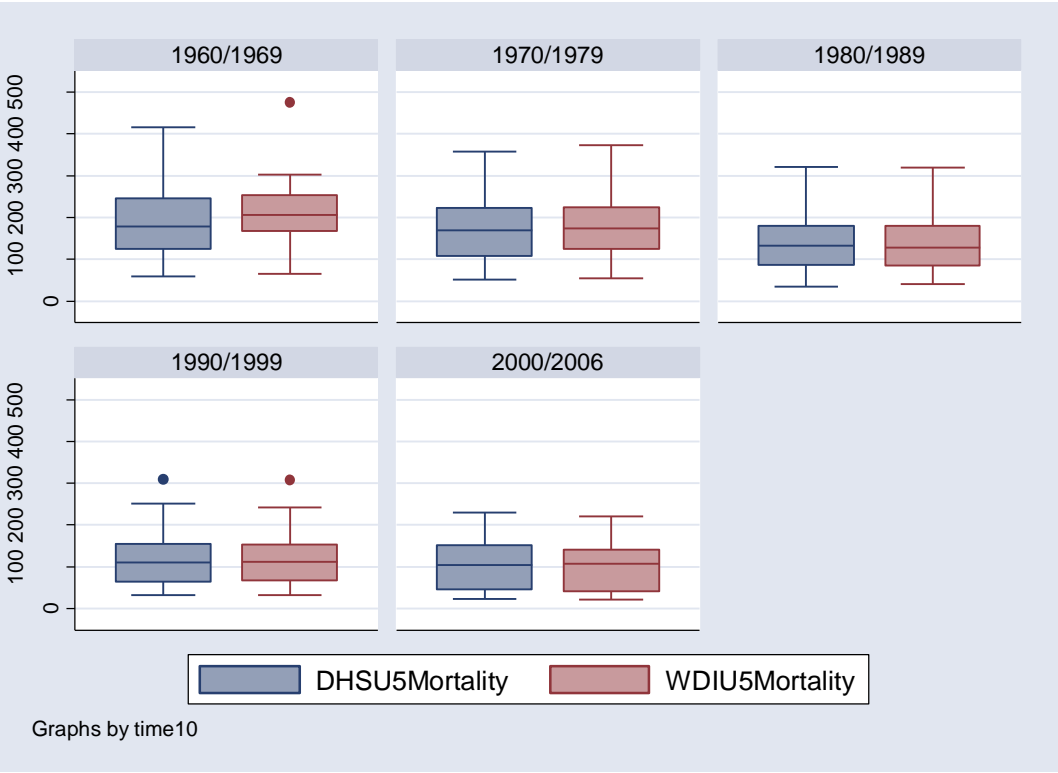
Table 1: t-test of difference in means between WDI and DHS datasets

		WDI			DHS					
	N	Mean	Standard Error	Stand deviation	Mean	Stand ard Error	Standa rd Deviation	T – value two tailed t test	P-value two tailed test	Means are significant ly different?
All years	252	142.50	4.60	73.06	139.43	4.62	73.28	1.53	0.13	No
1960/1969	22	210.35	17.79	83.48	183.84	19.06	89.40	4.76	0.0001	Yes
1970/1979	58	179.63	9.11	69.38	173.55	10.17	77.48	1.20	0.234	No
1980/1989	69	137.29	7.54	62.66	137.88	7.86	65.31	0.1751	0.8615	No
1990/1999	60	116.414	7.83	60.7	117.68	7.86	60.91	-0.34	0.7348	No
2000/2006	60	102.43	8.49	58.03	103.88	8.64	56.69	-0.3732	0.7109	No

Graph1: Comparison of DHS and WDI Under-five Mortality in all Decades



Graph 2: Comparison of DHS and WDI Under-five mortality by decade



Appendix 4: Regressions Showing Whether Countries are on Course to Achieve MDG4

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
CMR	Linear	Armenia	1995	2002	7500.57	-3.738263	-2.05	0.086	Yes
CMR	Linear	Bangladesh	1983	2001	9594	-4.758167	-12.99	0	Yes
CMR	Linear	Benin	1986	1998	7984.193	-3.925895	-3.62	0.004	No
CMR	Linear	Bolivia	1979	2000	6607.176	-3.263677	-7.53	0	Yes
CMR	Linear	Brazil	1976	1993	10131.32	-5.06188	-7.17	0	Yes
CMR	Linear	Burkina Faso	1982	2000	4971.196	-2.395654	-2.66	0.017	No
CMR	Linear	Burundi	1977	1984	30651.49	-15.38749	-7.1	0	Yes
CMR	Linear	Central African Republic	1984	1991	-65.7823	0.111607	0.16	0.881	No
CMR	Linear	Cambodia	1990	2002	3476.337	-1.687776	-1.35	0.205	No
CMR	Linear	Cameroon	1994	2001	43.18171	0.047313	0.07	0.943	No
CMR	Linear	Chad	1986	2001	2161.586	-0.989242	-0.68	0.51	No
CMR	Linear	Colombia	1976	1997	3228.443	-1.605683	-6.38	0	Yes
CMR	Linear	Comoros	1986	1993	5994.499	-2.961871	-0.68	0.519	No
CMR	Linear	Congo	1995	2002	3927.62	-1.906596	-0.81	0.45	No
CMR	Linear	Cote d'Ivoire	1984	2002	4176.169	-2.028524	-1.66	0.115	No
CMR	Linear	Dominican Republic	1976	1999	5356.576	-2.663449	-9.95	0	Yes
CMR	Linear	Ecuador	1977	1984	9367.213	-4.68866	-2.21	0.69	No
CMR	Linear	Egypt	1978	2002	11262.47	-5.61472	-16.56	0	Yes
CMR	Linear	Gabon	1990	1997	8340	-4.1421	-2.04	0.088	Yes
CMR	Linear	Ghana	1978	2000	5282.434	-2.594911	-5.55	0	No
CMR	Linear	Guatemala	1977	1994	10245.12	-5.113891	-10.43	0	Yes
CMR	Linear	Guinea	1989	2001	3749.02	-1.788302	-1.24	0.242	No
CMR	Linear	Guyana	1995	2001	5315.014	-2.639808	-0.79	0.465	No
CMR	Linear	Haiti	1995	2001	6722.702	-3.312814	-4.18	0.001	No
CMR	Linear	Honduras	1995	2001	1009.965	-0.4868555	-0.62	0.562	No

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
CMR	Linear	Indonesia	1977	1987	6743.032	-3.348175	-3.87	0.004	Yes
CMR	Linear	Kazakhstan	1985	1995	-9610.02	4.855161	3	0.015	No
CMR	Linear	Kenya	1979	1999	-2908.82	1.507972	4.27	0	No
CMR	Linear	Kyrgyz Republic	1987	1993	1380.504	-0.6564823	-0.15	0.884	No
CMR	Linear	Lesotho	1994	2000	-5432.17	2.765456	0.73	0.496	No
CMR	Linear	Liberia	1976	1982	6405.592	-3.122487	-1.95	0.108	No
CMR	Linear	Madagascar	1982	1999	11846.45	-5.877113	-7.43	0	Yes
CMR	Linear	Malawi	1982	2000	9565.131	-4.703284	-4.78	0	No
CMR	Linear	Mali	1977	1997	5730.308	-2.759445	-2.09	0.051	No
CMR	Linear	Mexico	1977	1984	10979.64	-5.509475	-4.17	0.006	Yes
CMR	Linear	Moldova	1994	2001	5986.267	-2.981268	-1.42	0.215	No
CMR	Linear	Morocco	1993	1999	7343.73	-3.652209	-8.1	0	Yes
CMR	Linear	Mozambique	1987	1999	13366.88	-6.602971	-5.14	0	Yes
CMR	Linear	Namibia	1982	1996	7849.122	-3.910978	-4.13	0.001	Yes
CMR	Linear	Nicaragua	1987	1997	5439.476	-2.704576	-5.86	0	Yes
CMR	Linear	Niger	1982	1994	-990.477	0.6533378	0.39	0.703	No
CMR	Linear	Nigeria	1980	1999	-842.598	0.5080056	0.35	0.727	No
CMR	Linear	Pakistan	1980	1986	9259.596	-4.608276	-1.6	0.17	No
CMR	Linear	Paraguay	1980	1986	1295.061	-0.6301915	-0.3	0.776	No
CMR	Linear	Peru	1976	1988	7569.008	-3.769288	-5.49	0	Yes
CMR	Linear	Philippines	1983	1999	5834.185	-2.905022	-8.95	0	Yes
CMR	Linear	Rwanda	1982	2001	-3185.83	1.687928	1.26	0.224	No
CMR	Linear	Senegal	1976	2001	6889.623	-3.387889	-6.46	0	No
CMR	Linear	Sri Lanka	1977	1983	4062	-2.030288	-1.12	0.313	No
CMR	Linear	Sudan	1980	1994	19076.06	-9.553795	-8.76	0	Yes
CMR	Linear	Thailand	1977	1983	3153.857	-1.569471	-0.7	0.514	No
CMR	Linear	Togo	1978	1993	2856.342	-1.368019	-1.7	0.116	No
CMR	Linear	Tanzania	1982	2000	2979.901	-1.424984	-1.53	0.144	No
CMR	Linear	Trinidad and Tobago	1977	1983	-1635.41	0.8426204	0.35	0.742	No

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
CMR	Linear	Turkey	1983	1994	9341.906	-4.662284	-6.34	0	Yes
CMR	Linear	Uganda	1978	1996	4611.789	-2.238943	-3.37	0.004	No
CMR	Linear	Uzbekistan	1986	1992	632.3806	-0.2920833	-0.15	0.883	No
CMR	Linear	Vietnam	1987	1998	6438	-3.210776	-4.39	0.001	Yes
CMR	Linear	Yemen	1981	1987	10540.25	-5.242497	-1.79	0.133	No
CMR	Linear	Zambia	1982	1997	1752.056	-0.7964937	-0.8	0.435	No
CMR	Linear	Zimbabwe	1978	2002	2786.969	-1.3644	-2.99	0.007	No
IMR	Linear	Armenia	1995	2004	3637.42	-1.805	-1.54	0.163	No
IMR	Linear	Bangladesh	1983	2003	6552.432	-3.243356	-10.56	0	Yes
IMR	Linear	Benin	1986	2000	4374.287	-2.142652	-3.29	0.006	No
IMR	Linear	Bolivia	1979	2002	6259.179	-3.100815	-10.33	0	Yes
IMR	Linear	Brazil	1976	1995	8760.865	-4.374864	-9.22	0	Yes
IMR	Linear	Burkina Faso	1982	2002	5965.703	-2.932435	-5.32	0	No
IMR	Linear	Burundi	1977	1986	16092.72	-8.066116	-7.27	0	Yes
IMR	Linear	Central African Republic	1984	1993	4812.002	-2.366806	-1.92	0.092	No
IMR	Linear	Cambodia	1990	2004	4914.51	-2.41708	-2.42	0.031	No
IMR	Linear	Cameroon	1994	2003	688.6677	-0.301857	-0.6	0.556	No
IMR	Linear	Chad	1986	2003	3916.728	-1.90017	-2.48	0.025	No
IMR	Linear	Colombia	1976	1999	2856.361	-1.420408	-7.57	0	Yes
IMR	Linear	Comoros	1986	1995	7729.691	-3.84245	-1.37	0.209	No
IMR	Linear	Congo	1995	2004	1944.419	-0.9301679	-0.55	0.594	No
IMR	Linear	Cote d'Ivoire	1984	2004	-2763.93	1.442207	1.11	0.289	No
IMR	Linear	Dominican Republic	1976	2001	4072.282	-2.021343	-10.83	0	Yes
IMR	Linear	Ecuador	1977	1986	10210.56	-5.119693	-4.03	0.004	Yes
IMR	Linear	Egypt	1978	2004	8678.304	-4.321951	-13.78	0	Yes
IMR	Linear	Gabon	1990	1999	5028.202	-2.489391	-2.02	0.078	Yes
IMR	Linear	Ghana	1978	2002	3766.794	-1.851846	-6.09	0	No
IMR	Linear	Guatemala	1977	1996	6927.321	-3.450251	-9.93	0	Yes

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
IMR	Linear	Guinea	1989	2003	7332.896	-3.6140448	-4.3	0.001	No
IMR	Linear	Guyana	1995	2003	116.1678	-0.0388065	-0.02	0.982	No
IMR	Linear	Haiti	1995	2003	5221.473	-2.577044	-5.39	0	Yes
IMR	Linear	Honduras	1995	2003	3581	-1.777091	-3.03	0.016	Yes
IMR	Linear	Indonesia	1977	1989	4211.024	-2.08261	-5.85	0	Yes
IMR	Linear	Kazakhstan	1985	1997	-4239.64	2.155468	1.73	0.109	No
IMR	Linear	Kenya	1979	2001	-1697.39	0.8885685	3.66	0.001	No
IMR	Linear	Kyrgyz Republic	1987	1995	3230.66	-1.58872	-0.86	0.413	No
IMR	Linear	Lesotho	1994	2002	-856.331	0.4690939	0.23	0.826	No
IMR	Linear	Liberia	1976	1984	4334.607	-2.0966	-1.38	0.211	No
IMR	Linear	Madagascar	1982	2001	6533.739	-3.229825	-5.13	0	Yes
IMR	Linear	Malawi	1982	2002	5810	-2.851137	-4.59	0	No
IMR	Linear	Mali	1977	1999	4250.485	-2.056593	-2.56	0.018	No
IMR	Linear	Mexico	1977	1986	10797.26	-5.420837	-5.79	0.001	Yes
IMR	Linear	Moldova	1994	2003	3728.585	-1.853686	-1.29	0.24	No
IMR	Linear	Morocco	1993	2001	5176.565	-2.56801	-8.56	0	Yes
IMR	Linear	Mozambique	1987	2001	10842	-5.360359	-5.46	0	Yes
IMR	Linear	Namibia	1982	1998	6252.659	-3.115153	-5.41	0	Yes
IMR	Linear	Nicaragua	1987	1999	4682.086	-2.327614	-9.55	0	Yes
IMR	Linear	Niger	1982	1996	-1276.74	0.721628	0.73	0.476	No
IMR	Linear	Nigeria	1980	2001	405.681	-0.15238	-0.27	0.793	No
IMR	Linear	Pakistan	1980	1988	3174	-1.545783	-0.81	0.445	No
IMR	Linear	Paraguay	1980	1988	1560.193	-0.7682799	-0.71	0.503	No
IMR	Linear	Peru	1976	1990	7642.002	-3.814606	-0.71	0.503	No
IMR	Linear	Philippines	1983	2001	3103.414	-1.539081	-5.21	0	Yes
IMR	Linear	Rwanda	1982	2003	-1171.75	0.64524	0.76	0.454	No
IMR	Linear	Senegal	1976	2003	4159.555	-2.043487	-5.43	0	No
IMR	Linear	Sri Lanka	1977	1985	4864	-2.4397	-1.96	0.091	Yes
IMR	Linear	Sudan	1980	1996	8843.639	-4.41	-3.76	0.007	Yes

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
IMR	Linear	Thailand	1977	1985	4726.747	-2.36707	-1.8	0.114	No
IMR	Linear	Togo	1978	1995	3392.33	-1.659819	-3.04	0.008	No
IMR	Linear	Tanzania	1982	2002	3614.187	-1.763507	-2.75	0.013	No
IMR	Linear	Trinidad and Tobago	1977	1985	-39.8839	0.0361652	0.03	0.98	No
IMR	Linear	Turkey	1983	1996	6192.866	-3.081167	5.05	0	Yes
IMR	Linear	Uganda	1978	1998	4381.418	-2.148646	-4.3	0	No
IMR	Linear	Uzbekistan	1986	1994	-180.34	0.1133186	0.09	0.928	No
IMR	Linear	Vietnam	1987	2000	4441.959	-2.211993	-4.21	0.001	Yes
IMR	Linear	Yemen	1981	1989	13974	-6.983783	-4.05	0.005	Yes
IMR	Linear	Zambia	1982	1999	-1308.08	0.7103335	1.38	0.186	No
IMR	Linear	Zimbabwe	1978	2004	1056.322	-0.5013329	-1.6	0.122	No
CMR	Lin-Log	Armenia	1995	2002	56837.15	-7474.522	-2.05	0.086	No
CMR	Lin-Log	Bangladesh	1983	2001	72126	-9478.886	-13	0	No
CMR	Lin-Log	Benin	1986	1998	59579.61	-7821.086	-3.62	0.004	No
CMR	Lin-Log	Bolivia	1979	2000	49429.97	-6492.662	-7.52	0	No
CMR	Lin-Log	Brazil	1976	1993	76347.98	-10043.56	-7.17	0	No
CMR	Lin-Log	Burkina Faso	1982	2000	36425.82	-4768.631	-2.66	0.017	No
CMR	Lin-Log	Burundi	1977	1984	231524.2	-30476.15	-7.11	0	No
CMR	Lin-Log	Central African Republic	1984	1991	-1531	222.1355	0.16	0.88	No
CMR	Lin-Log	Cambodia	1990	2002	25668.57	-3363.782	-1.34	0.206	No
CMR	Lin-Log	Cameroon	1994	2001	-579.077	94.3	0.07	0.943	No
CMR	Lin-Log	Chad	1986	2001	15151.03	-1969.229	-0.67	0.511	No
CMR	Lin-Log	Colombia	1976	1997	24272.19	-3191.078	-6.39	0	No
CMR	Lin-Log	Comoros	1986	1993	44837.63	-5889.667	-0.68	0.52	No
CMR	Lin-Log	Congo	1995	2002	29045.38	-3806.252	-0.81	0.45	No
CMR	Lin-Log	Cote d'Ivoire	1984	2002	30833.93	-4040.94	-1.66	0.115	No
CMR	Lin-Log	Dominican Republic	1976	1999	40270.31	-5294.182	-9.96	0	No
CMR	Lin-Log	Ecuador	1977	1984	70536.65	-9280.77	-2.21	0.069	No

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
CMR	Lin-Log	Egypt	1978	2002	84986.11	-11176.7	-16.63	0	No
CMR	Lin-Log	Gabon	1990	1997	62820.39	-8257.518	-2.04	0.088	No
CMR	Lin-Log	Ghana	1978	2000	39326.59	-5161.746	-5.55	0	No
CMR	Lin-Log	Guatemala	1977	1994	77196.47	-10153.91	-10.43	0	No
CMR	Lin-Log	Guinea	1989	2001	27281.32	-3566.537	-1.24	0.242	No
CMR	Lin-Log	Guyana	1995	2001	40112.4	-5272.663	-0.79	0.465	No
CMR	Lin-Log	Haiti	1995	2001	50263.5	-6600.059	-4.18	0.001	No
CMR	Lin-Log	Honduras	1995	2001	7423.263	-971.8594	-0.62	0.562	No
CMR	Lin-Log	Indonesia	1977	1987	50491.28	-6636.625	-3.87	0.004	No
CMR	Lin-Log	Kazakhstan	1985	1995	-73303.5	9657.234	3	0.015	Yes
CMR	Lin-Log	Kenya	1979	1999	-22684.9	2998.591	4.27	0	Yes
CMR	Lin-Log	Kyrgyz Republic	1987	1993	10002	-1307.025	-0.15	0.884	No
CMR	Lin-Log	Lesotho	1994	2000	-41848.7	5518.74	0.73	0.496	No
CMR	Lin-Log	Liberia	1976	1982	47127.07	-6179.017	-1.95	0.109	No
CMR	Lin-Log	Madagascar	1982	1999	88981.27	-11694.52	-7.42	0	No
CMR	Lin-Log	Malawi	1982	2000	71308	-9360	-4.78	0	No
CMR	Lin-Log	Mali	1977	1997	41914.19	-5486.547	-2.09	0.051	No
CMR	Lin-Log	Mexico	1977	1984	82887.61	-10910.07	-4.17	0.006	No
CMR	Lin-Log	Moldova	1994	2001	45281.96	-5954.323	-1.42	0.215	No
CMR	Lin-Log	Morocco	1993	1999	55257.03	-7264.618	-8.12	0	No
CMR	Lin-Log	Mozambique	1987	1999	100161.9	-13156.46	-5.13	0	No
CMR	Lin-Log	Namibia	1982	1996	59169.58	-7780.961	-4.13	0.001	No
CMR	Lin-Log	Nicaragua	1987	1997	40983.53	-5387.936	-5.86	0	No
CMR	Lin-Log	Niger	1982	1994	-9548.78	1297.865	0.39	0.703	No
CMR	Lin-Log	Nigeria	1980	1999	-7443.86	1002.147	0.35	0.73	No
CMR	Lin-Log	Pakistan	1980	1986	69511.67	-9139.481	-1.6	0.17	No
CMR	Lin-Log	Paraguay	1980	1986	9541.595	-1250.757	-0.3	0.775	No
CMR	Lin-Log	Peru	1976	1988	56806.81	-7469.649	-5.49	0	No
CMR	Lin-Log	Philippines	1983	1999	43990.91	-5784.41	-8.95	0	No

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
CMR	Lin-Log	Rwanda	1982	2001	-25409	3367.892	1.27	0.223	No
CMR	Lin-Log	Senegal	1976	2001	51362.24	-6742.405	-6.48	0	No
CMR	Lin-Log	Sri Lanka	1977	1983	30545.84	-4018.473	-1.12	0.313	No
CMR	Lin-Log	Sudan	1980	1994	144284	-18986.63	-8.77	0	No
CMR	Lin-Log	Thailand	1977	1983	23629.84	-3106.836	-0.7	0.514	No
CMR	Lin-Log	Togo	1978	1993	20762.63	-2715.764	-1.7	0.116	No
CMR	Lin-Log	Tanzania	1982	2000	21700.95	-2837.964	-1.54	0.144	No
CMR	Lin-Log	Trinidad and Tobago	1977	1983	-12635.9	1668.965	0.35	0.742	No
CMR	Lin-Log	Turkey	1983	1994	70493	-9272	-6.34	0	No
CMR	Lin-Log	Uganda	1978	1996	33949.29	-4448.854	-3.37	0.004	No
CMR	Lin-Log	Uzbekistan	1986	1992	4480.582	-583.1375	-0.15	0.883	No
CMR	Lin-Log	Vietnam	1987	1998	48648.63	-6398.192	-4.4	0.001	No
CMR	Lin-Log	Yemen	1981	1987	79110.34	-10400.71	-1.79	0.133	No
CMR	Lin-Log	Zambia	1982	1997	12161	-1579.011	-0.8	0.437	No
CMR	Lin-Log	Zimbabwe	1978	2002	20711.26	-2717.189	-2.99	0.006	No
IMR	Lin-Log	Armenia	1995	2004	27487.78	-3612.812	-1.54	0.163	No
IMR	Lin-Log	Bangladesh	1983	2003	49211.12	-6465.732	-10.58	0	No
IMR	Lin-Log	Benin	1986	2000	32545.33	-4270.063	-3.29	0.006	No
IMR	Lin-Log	Bolivia	1979	2002	46971.3	-6172.125	-10.33	0	No
IMR	Lin-Log	Brazil	1976	1995	66031.11	-8685.782	-9.21	0	No
IMR	Lin-Log	Burkina Faso	1982	2002	44495.14	-5840.659	-5.31	0	No
IMR	Lin-Log	Burundi	1977	1986	121428.1	-15980.59	-7.26	0	No
IMR	Lin-Log	Central African Republic	1984	1993	35832.61	-4703.932	-1.91	0.092	No
IMR	Lin-Log	Cambodia	1990	2004	36727.51	-4821.429	-2.42	0.031	No
IMR	Lin-Log	Cameroon	1994	2003	4660.862	-602.022	-0.6	0.555	No
IMR	Lin-Log	Chad	1986	2003	28912.01	-3788.407	-2.48	0.025	No
IMR	Lin-Log	Colombia	1976	1999	21487.14	-2824.871	-7.59	0	No
IMR	Lin-Log	Comoros	1986	1995	58142.31	-7643.49	-1.37	0.209	No

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
IMR	Lin-Log	Congo	1995	2004	14206.56	-1858	-0.55	0.595	No
IMR	Lin-Log	Cote d'Ivoire	1984	2004	-21691	2869.58	1.11	0.289	No
IMR	Lin-Log	Dominican Republic	1976	2001	30584.53	-4019.943	-10.84	0	No
IMR	Lin-Log	Ecuador	1977	1986	77064.79	-10142.63	-4.02	0.004	No
IMR	Lin-Log	Egypt	1978	2004	65470.7	-8609.017	-13.84	0	No
IMR	Lin-Log	Gabon	1990	1999	37800.42	-4966.646	-2.02	0.078	No
IMR	Lin-Log	Ghana	1978	2002	28072.95	-3685.065	-6.09	0	No
IMR	Lin-Log	Guatemala	1977	1996	52146.78	-6857.061	-9.94	0	No
IMR	Lin-Log	Guinea	1989	2003	54924.37	-7212.245	-4.29	0.001	No
IMR	Lin-Log	Guyana	1995	2003	630.1637	-77.83405	-0.02	0.982	No
IMR	Lin-Log	Haiti	1995	2003	39125	-5138.55	-5.39	0	No
IMR	Lin-Log	Honduras	1995	2003	27021.26	-3551.476	-3.03	0.016	No
IMR	Lin-Log	Indonesia	1977	1989	31448.77	-4131.462	-5.85	0	No
IMR	Lin-Log	Kazakhstan	1985	1997	-32561.5	4293.276	1.73	0.109	No
IMR	Lin-Log	Kenya	1979	2001	-13363	1768.575	3.66	0.001	Yes
IMR	Lin-Log	Kyrgyz Republic	1987	1995	24101.56	-3163.875	-0.86	0.413	No
IMR	Lin-Log	Lesotho	1994	2002	-7027.37	935.3129	0.23	0.827	No
IMR	Lin-Log	Liberia	1976	1984	31674.68	-4148.591	-1.38	0.211	No
IMR	Lin-Log	Madagascar	1982	2001	48943	-6429.361	-5.12	0	No
IMR	Lin-Log	Malawi	1982	2002	43245.95	-5675.275	-4.58	0	No
IMR	Lin-Log	Mali	1977	1999	31234.42	-4091.236	-2.56	0.018	No
IMR	Lin-Log	Mexico	1977	1986	81576.12	-10738.21	-5.79	0.001	No
IMR	Lin-Log	Moldova	1994	2003	28169.3	-3703.26	-1.28	0.24	No
IMR	Lin-Log	Morocco	1993	2001	38895.37	-5112	-8.58	0	No
IMR	Lin-Log	Mozambique	1987	2001	81349.08	-10686.48	-5.46	0	No
IMR	Lin-Log	Namibia	1982	1998	47144.26	-6199.508	-5.41	0	No
IMR	Lin-Log	Nicaragua	1987	1999	35288.83	-4639.179	-9.55	0	No
IMR	Lin-Log	Niger	1982	1996	-10740.8	1434.999	0.73	0.476	No
IMR	Lin-Log	Nigeria	1980	2001	2427.93	-306.1502	-0.27	0.791	No

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
IMR	Lin-Log	Pakistan	1980	1988	23412.22	-3069.255	-0.81	0.445	No
IMR	Lin-Log	Paraguay	1980	1988	11612.42	-1524.653	-0.71	0.503	No
IMR	Lin-Log	Peru	1976	1990	57497.59	-7562.855	-6.14	0	No
IMR	Lin-Log	Philippines	1983	2001	23339.18	-3067.257	-5.22	0	No
IMR	Lin-Log	Rwanda	1982	2003	-9691.33	1290.646	0.77	0.452	No
IMR	Lin-Log	Senegal	1976	2003	30997	-4068.5	-5.44	0	No
IMR	Lin-Log	Sri Lanka	1977	1985	36717.98	-4832.55	-1.96	0.091	No
IMR	Lin-Log	Sudan	1980	1996	66536.86	-8750.829	-3.76	0.007	No
IMR	Lin-Log	Thailand	1977	1985	35629.62	-4688.496	-1.8	0.114	No
IMR	Lin-Log	Togo	1978	1995	25134.39	-3297.193	-3.04	0.008	No
IMR	Lin-Log	Tanzania	1982	2002	26774.1	-3511.018	-2.75	0.013	No
IMR	Lin-Log	Trinidad and Tobago	1977	1985	-515.929	72.14628	0.03	0.98	No
IMR	Lin-Log	Turkey	1983	1996	46641.49	-6132.284	-5.06	0	No
IMR	Lin-Log	Uganda	1978	1998	32545.58	-4270.729	-4.3	0	No
IMR	Lin-Log	Uzbekistan	1986	1994	-1652.8	223.5366	0.09	0.929	No
IMR	Lin-Log	Vietnam	1987	2000	33544.48	-4410.857	-4.21	0.001	No
IMR	Lin-Log	Yemen	1981	1989	105361.7	-13860.76	-4.05	0.005	No
IMR	Lin-Log	Zambia	1982	1999	-10660.6	1417.356	1.39	0.185	No
IMR	Lin-Log	Zimbabwe	1978	2004	7656.22	-1000.219	-1.61	0.121	No
CMR	Log - Lin	Armenia	1995	2002	237.3518	-0.1171354	-1.33	0.231	No
CMR	Log - Lin	Bangladesh	1983	2001	88.22527	-0.0419181	-12.48	0	No
CMR	Log - Lin	Benin	1986	1998	52.0558	-0.0235764	-3.68	0.004	No
CMR	Log - Lin	Bolivia	1979	2000	63.3687	-0.0294823	-7.27	0	No
CMR	Log - Lin	Brazil	1976	1993	134.737	-0.0656853	-7.3	0	No
CMR	Log - Lin	Burkina Faso	1982	2000	28.98694	-0.0118978	-2.7	0.015	No
CMR	Log - Lin	Burundi	1977	1984	178.1957	-0.087374	-7.74	0	No
CMR	Log - Lin	Central African Republic	1984	1991	3.799	0.000629	0.14	0.895	No
CMR	Log - Lin	Cambodia	1990	2002	47.363	-0.02139	-1.5	0.161	No

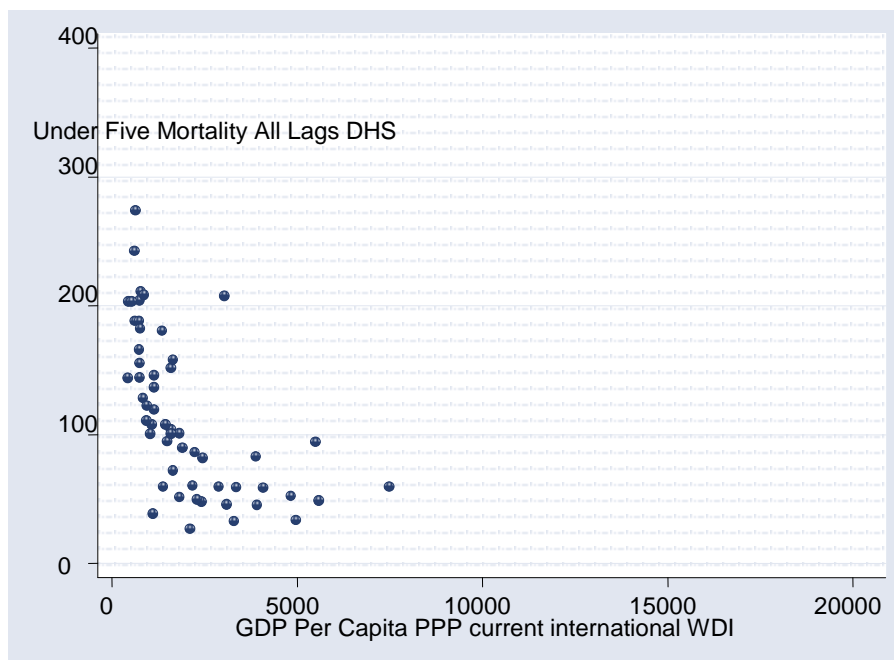
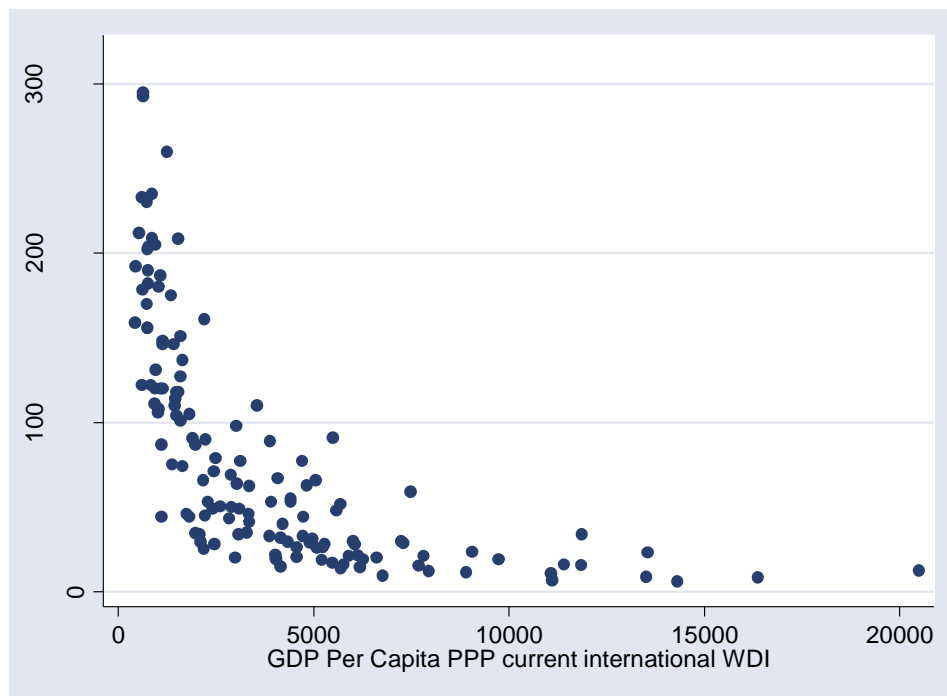
Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
CMR	Log - Lin	Cameroon	1994	2001	3.894	0.0005129	0.1	0.919	No
CMR	Log - Lin	Chad	1986	2001	17.306	-0.006055	-0.79	0.442	No
CMR	Log - Lin	Colombia	1976	1997	85.13429	-0.0410414	-6.07	0	No
CMR	Log - Lin	Comoros	1986	1993	52.76	-0.0242116	-0.56	0.597	No
CMR	Log - Lin	Congo	1995	2002	40.73	-0.01799	-0.91	0.399	No
CMR	Log - Lin	Cote d'Ivoire	1984	2002	39.74305	-0.0174996	-1.84	0.083	No
CMR	Log - Lin	Dominican Republic	1976	1999	91.947	-0.0442063	-9.92	0	No
CMR	Log - Lin	Ecuador	1977	1984	111.85	-0.0542	-2.2	0.07	No
CMR	Log - Lin	Egypt	1978	2002	138.5	-0.0674	-23.08	0	No
CMR	Log - Lin	Gabon	1990	1997	104.461	-0.050194	-2.1	0.081	No
CMR	Log - Lin	Ghana	1978	2000	48.73	-0.0221	-5.29	0	No
CMR	Log - Lin	Guatemala	1977	1994	122.5895	-0.0594947	-8.81	0	No
CMR	Log - Lin	Guinea	1989	2001	25.20519	-0.0100303	-1.24	0.239	No
CMR	Log - Lin	Guyana	1995	2001	186.45	-0.0915121	-0.89	0.413	No
CMR	Log - Lin	Haiti	1995	2001	62.446	-0.0289	-4.07	0.002	No
CMR	Log - Lin	Honduras	1995	2001	32.47	-0.0144	-0.69	0.52	No
CMR	Log - Lin	Indonesia	1977	1987	67.019	-0.0314603	-3.78	0.004	No
CMR	Log - Lin	Kazakhstan	1985	1995	-173.74	0.0892	2.82	0.02	No
CMR	Log - Lin	Kenya	1979	1999	-28.54	0.0166	4.17	0.001	No
CMR	Log - Lin	Kyrgyz Republic	1987	1993	11.22297	-0.003497	-0.06	0.955	No
CMR	Log - Lin	Lesotho	1994	2000	-69.3	0.036949	0.83	0.443	No
CMR	Log - Lin	Liberia	1976	1982	32.74	-0.0138	-1.96	0.107	No
CMR	Log - Lin	Madagascar	1982	1999	94.3777	-0.0449	-6.2	0	No
CMR	Log - Lin	Malawi	1982	2000	53.75	-0.0243	-5.07	0	No
CMR	Log - Lin	Mali	1977	1997	25.78	-0.0102	-1.64	0.0119	No
CMR	Log - Lin	Mexico	1977	1984	172.33	-0.0848903	-4.58	0.004	No
CMR	Log - Lin	Moldova	1994	2001	290.0407	-0.14351	-1.72	0.146	No
CMR	Log - Lin	Morocco	1993	1999	91.95847	-0.0440638	-8.58	0	No
CMR	Log - Lin	Mozambique	1987	1999	72.5	-0.0337078	-4.87	0	No

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
CMR	Log - Lin	Namibia	1982	1996	114.6918	-0.0555537	-3.69	0.002	No
CMR	Log - Lin	Nicaragua	1987	1997	108.0173	-0.0522	-5.73	0	No
CMR	Log - Lin	Niger	1982	1994	1.574	0.002	0.39	0.706	No
CMR	Log - Lin	Nigeria	1980	1999	1.369	0.001875	0.2	0.84	No
CMR	Log - Lin	Pakistan	1980	1986	77.60563	-0.0367195	-1.61	0.168	No
CMR	Log - Lin	Paraguay	1980	1986	29.37	-0.0129	-0.29	0.782	No
CMR	Log - Lin	Peru	1976	1988	83.72153	-0.0399337	-5.35	0	No
CMR	Log - Lin	Philippines	1983	1999	123.75	-0.0602	-8.18	0	No
CMR	Log - Lin	Rwanda	1982	2001	-12.8	0.0090194	1.14	0.269	No
CMR	Log - Lin	Senegal	1976	2001	47.516	-0.02138	-6.23	0	No
CMR	Log - Lin	Sri Lanka	1977	1983	110.3458	-0.053835	-1.15	0.303	No
CMR	Log - Lin	Sudan	1980	1994	221.7993	-0.1094111	-7.66	0	No
CMR	Log - Lin	Thailand	1977	1983	100.928	-0.0490515	-0.95	0.386	No
CMR	Log - Lin	Togo	1978	1993	23.94	-0.00957	-1.68	0.122	No
CMR	Log - Lin	Tanzania	1982	2000	24.95	-0.01005	0.153	0.153	No
CMR	Log - Lin	Trinidad and Tobago	1977	1983	-20.0262	0.011854	0.14	0.897	No
CMR	Log - Lin	Turkey	1983	1994	137.597	-0.067	-6.3	0	No
CMR	Log - Lin	Uganda	1978	1996	31.84429	-0.0134662	-3.43	0.003	No
CMR	Log - Lin	Uzbekistan	1986	1992	13.78	-0.0049	-0.12	0.907	No
CMR	Log - Lin	Vietnam	1987	1998	161.4969	-0.07922	-4.24	0.002	No
CMR	Log - Lin	Yemen	1981	1987	81.73572	-0.0387136	-1.81	0.13	No
CMR	Log - Lin	Zambia	1982	1997	15.16	-0.00505	-0.87	0.402	No
CMR	Log - Lin	Zimbabwe	1978	2002	42.15	-0.0190518	-2.92	0.008	No
IMR	Log - Lin	Armenia	1995	2004	99.57	-0.04818	-0.85	0.419	No
IMR	Log - Lin	Bangladesh	1983	2003	76.91742	-0.036364	-11.33	0	No
IMR	Log - Lin	Benin	1986	2000	46.247	-0.02088	-3.28	0.006	No
IMR	Log - Lin	Bolivia	1979	2002	79.43747	-0.0377	-9.45	0	No
IMR	Log - Lin	Brazil	1976	1995	133.0136	-0.06486	-9.25	0	No
IMR	Log - Lin	Burkina Faso	1982	2002	52.8535	-0.0241	-5.48	0	No

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
IMR	Log - Lin	Burundi	1977	1986	162.4711	-0.0796	-6.17	0	No
IMR	Log - Lin	Central African Republic	1984	1993	53.96937	-0.0248011	-1.99	0.082	No
IMR	Log - Lin	Cambodia	1990	2004	73.55903	-0.03461	-2.57	0.023	No
IMR	Log - Lin	Cameroon	1994	2003	10.415	-0.002993	-0.49	0.627	No
IMR	Log - Lin	Chad	1986	2003	36.73967	-0.0159969	-2.56	0.021	No
IMR	Log - Lin	Colombia	1976	1999	85.2415	-0.04112	-7.13	0	No
IMR	Log - Lin	Comoros	1986	1995	103.7425	-0.0499336	-1.47	0.18	No
IMR	Log - Lin	Congo	1995	2004	26.74	-0.01116	-0.58	0.58	No
IMR	Log - Lin	Cote d'Ivoire	1984	2004	-19.9918	0.01238	1.2	0.329	No
IMR	Log - Lin	Dominican Republic	1976	2001	83.39	-0.0399	-10.6	0	No
IMR	Log - Lin	Ecuador	1977	1986	177.56	-0.0875	-3.84	0.005	No
IMR	Log - Lin	Egypt	1978	2004	123.7959	-0.0600811	-21.33	0	No
IMR	Log - Lin	Gabon	1990	1999	81.28	-0.0386857	-1.96	0.085	No
IMR	Log - Lin	Ghana	1978	2002	52.017	-0.0239395	-10.37	0	No
IMR	Log - Lin	Guatemala	1977	1996	100.896	-0.04865	-10.37	0	No
IMR	Log - Lin	Guinea	1989	2003	70.74	-0.03305	-4.36	0.001	No
IMR	Log - Lin	Guyana	1995	2003	6.31	-0.00137	-0.03	0.98	No
IMR	Log - Lin	Haiti	1995	2003	69.37	-0.0325932	-5.44	0	No
IMR	Log - Lin	Honduras	1995	2003	152.4037	-0.0745777	-3.29	0.011	No
IMR	Log - Lin	Indonesia	1977	1989	55.58	-0.0258171	-5.5	0	No
IMR	Log - Lin	Kazakhstan	1985	1997	-82.431	0.04335	1.79	0.098	No
IMR	Log - Lin	Kenya	1979	2001	-20.444	0.0124094	3.59	0.002	No
IMR	Log - Lin	Kyrgyz Republic	1987	1995	43.76	-0.0198773	-0.73	0.483	No
IMR	Log - Lin	Lesotho	1994	2002	-9.688	0.00704	0.26	0.798	No
IMR	Log - Lin	Liberia	1976	1984	28.76	-0.0118951	-1.41	0.201	No
IMR	Log - Lin	Madagascar	1982	2001	77.9648	-0.0368472	-4.46	0	No
IMR	Log - Lin	Malawi	1982	2002	50.866	-0.0230945	-4.93	0	No
IMR	Log - Lin	Mali	1977	1999	27.67208	-0.01136	-2.04	0.054	No

Mortality	Functional Form	Country	Start Year	Finish Year	Intercept	Coefficient	t-statistic	Sig Level	Achieve MDG4 (P < 0.10)
IMR	Log - Lin	Mexico	1977	1986	193.85	-0.0958193	-6.75	0	No
IMR	Log - Lin	Moldova	1994	2003	200.11	-0.098595	-1.56	0.163	No
IMR	Log - Lin	Morocco	1993	2001	79.43754	-0.0378392	-9.23	0	No
IMR	Log - Lin	Mozambique	1987	2001	76.37	0.03557844	-5.71	0	No
IMR	Log - Lin	Namibia	1982	1998	121.1014	-0.058852	-5.82	0	No
IMR	Log - Lin	Nicaragua	1987	1999	112.96	-0.0547993	-9.18	0	No
IMR	Log - Lin	Niger	1982	1996	-4.312	0.0047	0.75	0.467	No
IMR	Log - Lin	Nigeria	1980	2001	7.889	-0.00164	-0.28	0.783	No
IMR	Log - Lin	Pakistan	1980	1988	28.95068	-0.0122371	-0.7	0.507	No
IMR	Log - Lin	Paraguay	1980	1988	48.97	-0.02289	-0.72	0.495	No
IMR	Log - Lin	Peru	1976	1990	108.44	-0.0525102	-6.16	0	No
IMR	Log - Lin	Philippines	1983	2001	83.83	-0.04033	-4.95	0	No
IMR	Log - Lin	Rwanda	1982	2003	-5.278	0.0050143	0.69	0.5	No
IMR	Log - Lin	Senegal	1976	2003	47.48486	-0.0215983	-5.43	0	No
IMR	Log - Lin	Sri Lanka	1977	1985	165.2896	-0.0817168	-1.65	0.143	No
IMR	Log - Lin	Sudan	1980	1996	100.105	-0.048178	-3.73	0.007	No
IMR	Log - Lin	Thailand	1977	1985	142.1298	-0.0699369	-2.12	0.071	No
IMR	Log - Lin	Togo	1978	1995	40.03	-0.01786	-3.21	0.005	No
IMR	Log - Lin	Tanzania	1982	2002	43.08447	-0.0193212	-2.89	0.009	No
IMR	Log - Lin	Trinidad and Tobago	1977	1985	0.2646	0.00158	0.03	0.979	No
IMR	Log - Lin	Turkey	1983	1996	100.128	-0.04826	-4.93	0	No
IMR	Log - Lin	Uganda	1978	1998	43.286	-0.0194168	-4.35	0	No
IMR	Log - Lin	Uzbekistan	1986	1994	-4.515	0.0041748	0.15	0.886	No
IMR	Log - Lin	Vietnam	1987	2000	134.52	-0.0657663	-3.63	0.003	No
IMR	Log - Lin	Yemen	1981	1989	134.9829	-0.0656352	-4.2	0.004	No
IMR	Log - Lin	Zambia	1982	1999	-10.46	0.0075964	1.48	0.158	No
IMR	Log - Lin	Zimbabwe	1978	2004	20.7662	-0.00841	-1.42	0.17	No

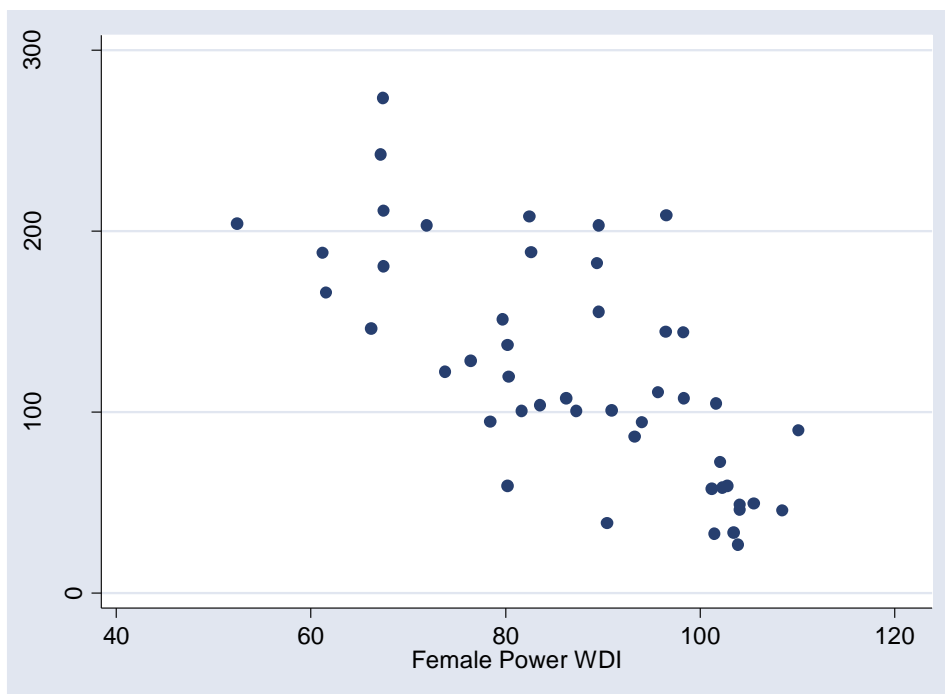
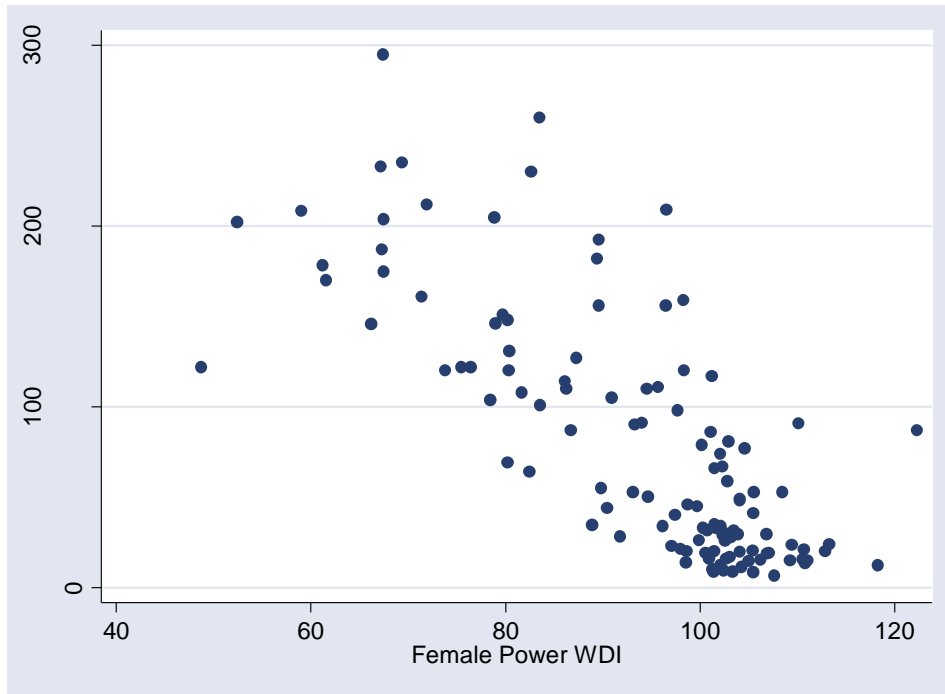
Appendix 5: Income and Under-five Mortality

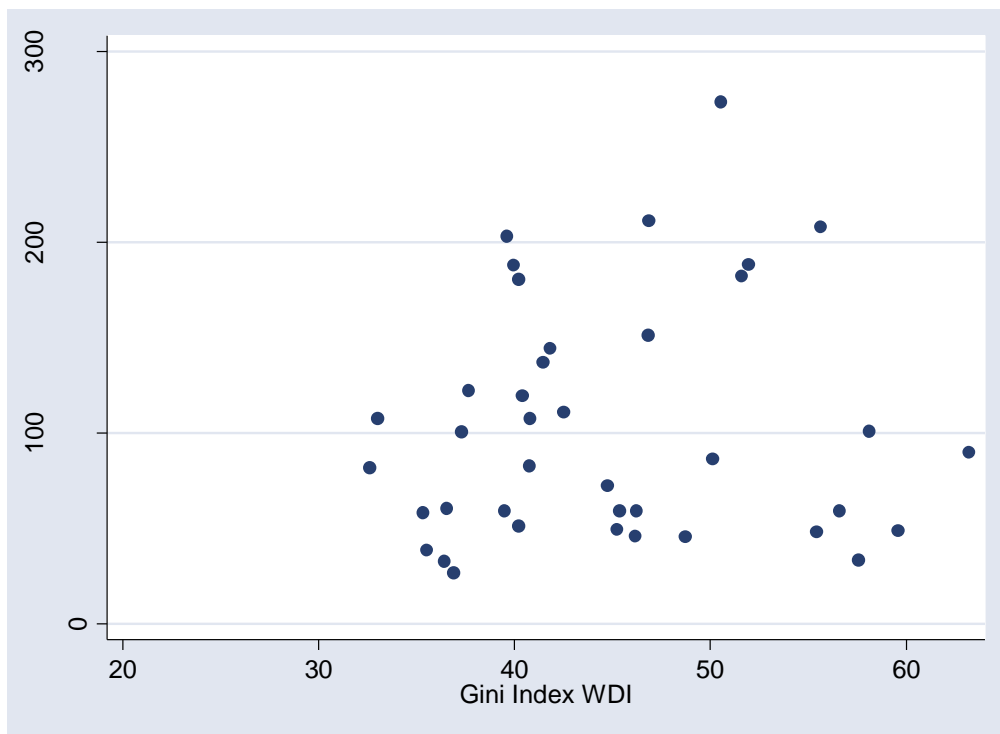
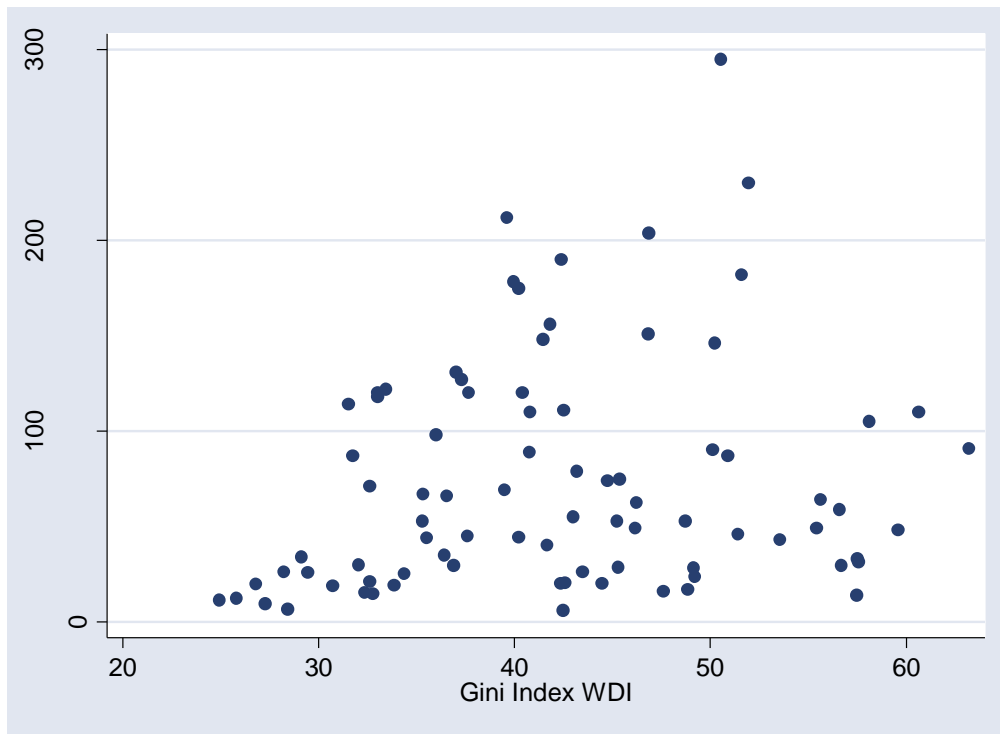


Appendix 6: Regression of Child Health and Income

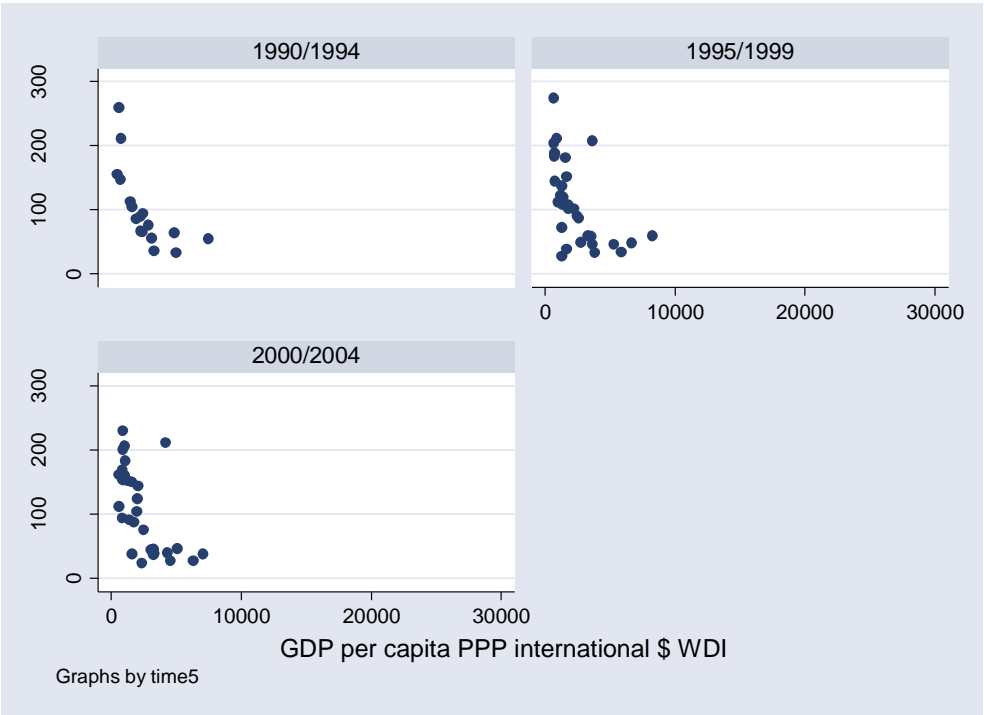
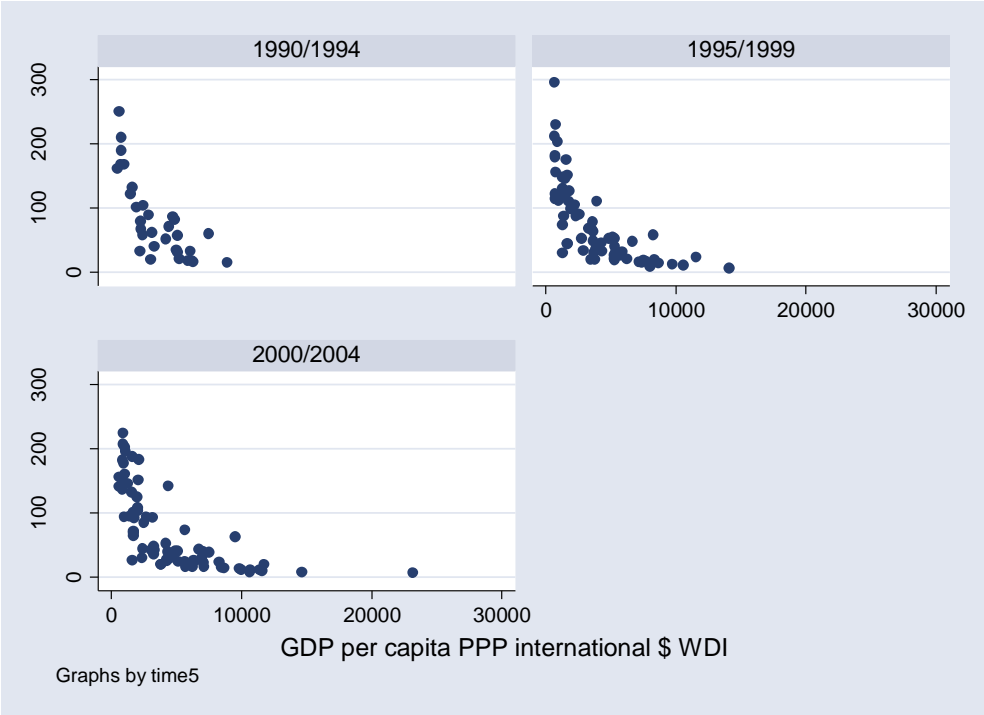
All results were estimated using OLS and the log-log model specification with no lag, for the period 1995 to 1999							
	Constant	Coefficient	N	t-statistic	P-value	Adjusted R-squared	Robust SE used?
Malnutrition prevalence weight for age (WDI)	7.698	-0.647	98	-7.11	0.000	0.375	Yes
Infant Mortality (WDI)	9.994	-0.783	141	-21.66	0.000	0.750	Yes
Under-five Mortality (WDI)	11.09	-0.882	141	-21.21	0.000	0.762	No
Neonatal Mortality (DHS)	5.826	-0.313	53	-4.94	0.000	0.31	Yes
Post Neonatal Mortality (DHS)	7.562	-0.548	53	-6.64	0.000	0.445	No
Infant Mortality (DHS)	7.423	-0.433	53	-6.61	0.000	0.446	No
Child Mortality (DHS)	10.563	-0.955	53	-7.14	0.000	0.495	No
Under-five Mortality (WDI)	8.947	-0.584	53	-7.24	0.000	0.492	No

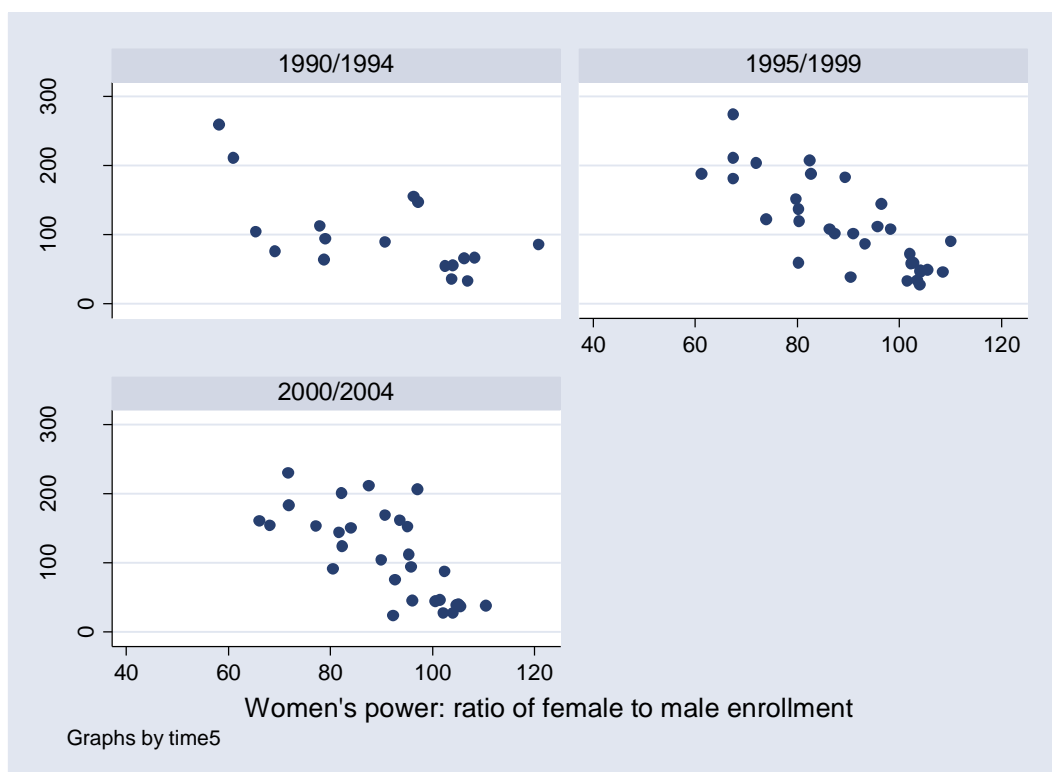
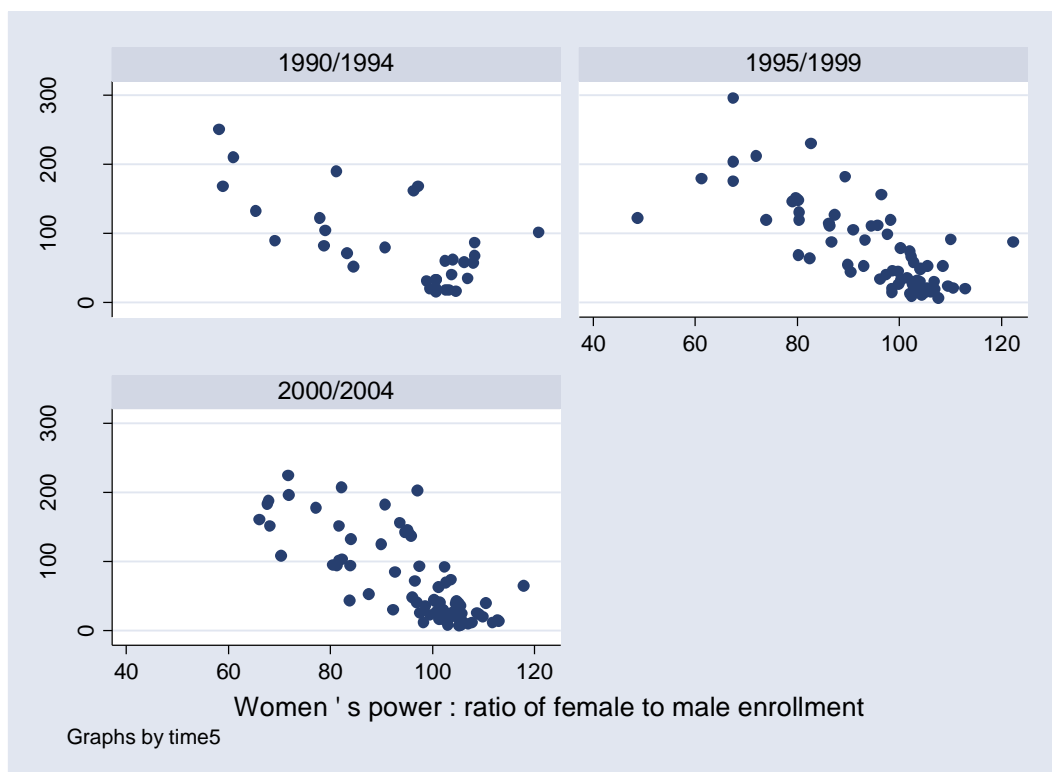
Appendix 7: Graphs of Income Inequality, Women's Power and Mortality

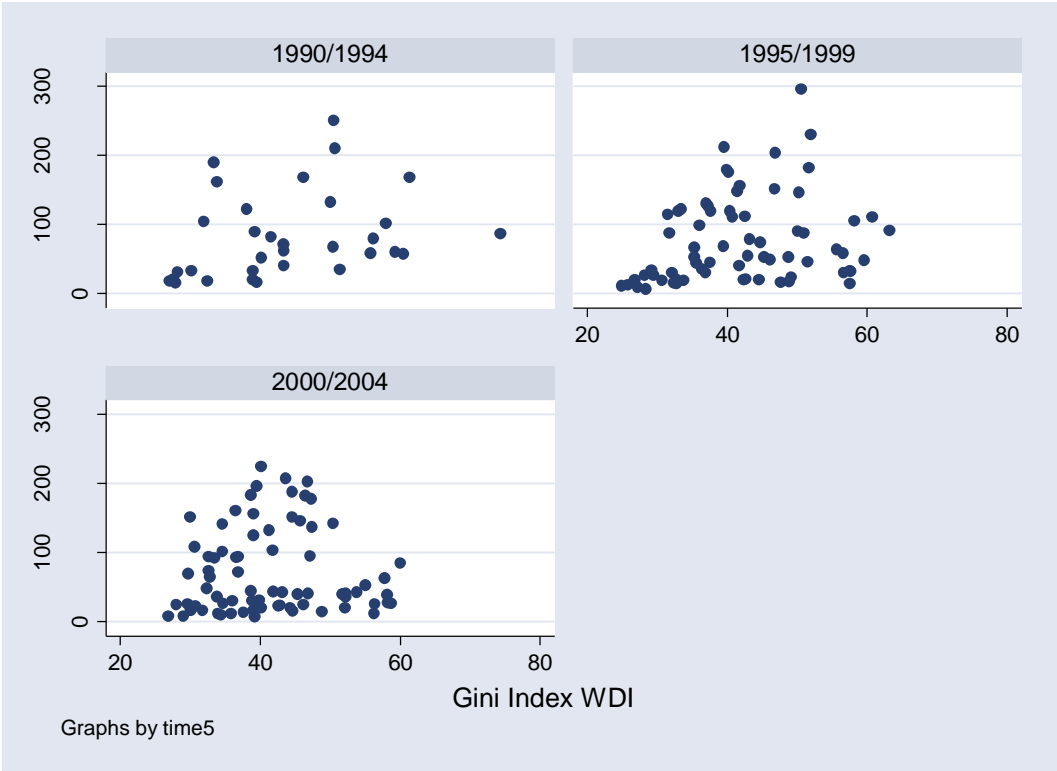




Appendix 8: Graphs of Income, Income Inequality and Women’s Power over Time







Appendix 9: XTFISHER Test of Stationarity

Dataset	Variable	Chi-squared	Prob>chi 2	Test demonstrates:
WDI	Malnutrition Prevalence, Weight for Age	315.797	0	Stationary
WDI	Infant Mortality	3079.3243	0	Stationary
WDI	Under-five Mortality	2812.2785	0	Stationary
DHS	Neonatal Mortality	363.0425	0	Stationary
DHS	Post Neonatal Mortality	212.7	0.0001	Stationary
DHS	Infant Mortality	150.19441	0.3	Not Stationary
DHS	Child Mortality	629.1611	0	Stationary
DHS	Under-five Mortality	330.7574	0	Stationary
DHS Adj	Child Mortality	182.2	0	Stationary
DHS Adj	Infant Mortality	190.8	0	Stationary
WDI	GDP per Capita in PPP	146.9019	1	Not stationary
WDI	Ratio of Girls to Boys in Primary and Secondary Education	1046.377	0	Stationary
WDI	Gini Index	181.0213	0	Stationary
DHS	Log Malnutrition Prevalence, Weight for Age	204.0145	0	Stationary
DHS	Log Infant Mortality	735.7108	0	Stationary
DHS	Log Under-five Mortality	732.0608	0	Stationary
DHS	Log Neonatal Mortality	256.6863	0	Stationary
DHS	Log Post Neonatal Mortality	108.9508	0.982	Non-stationary
DHS	Log Infant Mortality	86.7022	0.999	Non-stationary
DHS	Log Child Mortality	432.0515	0	Stationary
DHS	Log Under-five Mortality	152.3684	0.261	Non-stationary
DHS Adj	Log Child Mortality	55.6683	0.9954	Non-stationary
DHS Adj	Log Infant Mortality	101.745	0.4325	Non-stationary
WDI	Log GDP per Capita in PPP	1452.911	0	Stationary
WDI	Log Ratio of Girls to Boys in Primary and Secondary Education	960.78	0	Stationary
WDI	Log Gini Index	241.8	0	Stationary

Appendix 10: GLS Results accounting for AR(1)

GLS regression with natural logs taken of all variables and no lags										
	WDI	WDI	WDI	DHS	DHS	DHS	DHS	DHS	DHS Adj	DHS Adj
	Nutrition	Infant	Under-five	Neonatal	Post Neonatal	Infant	Child	Under-five	Child	Infant
GDP Per Capita, PPP (current US\$)	-0.950***	-0.395***	-0.675***	-0.445	-0.595***	-0.441***	-0.0540	-0.537***	-0.609***	-0.326**
Gini Index	-0.136	0.132	0.795***	-0.327	1.158***	0.906***	-0.492	0.587	0.783	0.537
Ratio of girls to boys in primary and secondary education	-0.558	-0.499*	-0.989***	-0.813	-1.029*	-1.139***	-0.894*	-0.852**	-1.134*	-1.274**
1989/1994Time dummy	0.119	0.110	0.0110	0.0403	-0.105	-0.102	0.203	-0.0135	0.0663	0.0844
2000/2004Time dummy	0.127	-0.157	-0.0898	-0.0561	-0.0784	-0.0361	-0.324*	-0.129	-0.0590	-0.107
Constant	13.02***	8.286***	10.83***	11.26***	8.039***	9.104***	8.622**	10.01***	11.12***	10.31***
Observations	82	142	142	63	65	65	65	65	39	47
Key										
* p<0.05										
** p<0.01										
*** p<0.001"										

GLS regression with natural logs of income only and no lags on any variable										
	WDI	WDI	WDI	DHS	DHS	DHS	DHS	DHS	DHS Adj	DHS Adj
	Nutrition	Infant	Under-five	Neonatal	Post Neonatal	Infant	Child	Under-five	Child	Infant
GDP Per Capita, PPP (current US\$)	-10.26***	-30.94***	-54.20***	-7.910*	-22.97**	-25.02***	-25.55***	-49.39***	-53.96***	-21.93***
Gini Index	0.0547	0.142	0.609*	0.119	-0.150	1.130***	1.242***	3.030***	2.902***	0.893*
Ratio of girls to boys in primary and secondary education	-0.316**	-0.496***	-0.998***	-0.395**	0.0249	-0.891***	-1.297***	-2.306***	-1.911***	-0.910***
1995/1999Time dummy	-1.405	-4.078	-2.723	0.335	-3.731	5.797	4.991	11.01	3.046	-4.323
2000/2004Time dummy	0.247	-9.351	-8.416	-1.202	-11.50	3.790	9.037	19.34	12.89	-13.21*
Constant	127.0***	314.2***	551.7***	124.3***	180.6**	280.6***	292.0***	538.2***	539.1***	285.3***
Observations	82	142	142	63	65	65	65	65	39	47
Key										
* p<0.05										
** p<0.01										
*** p<0.001"										

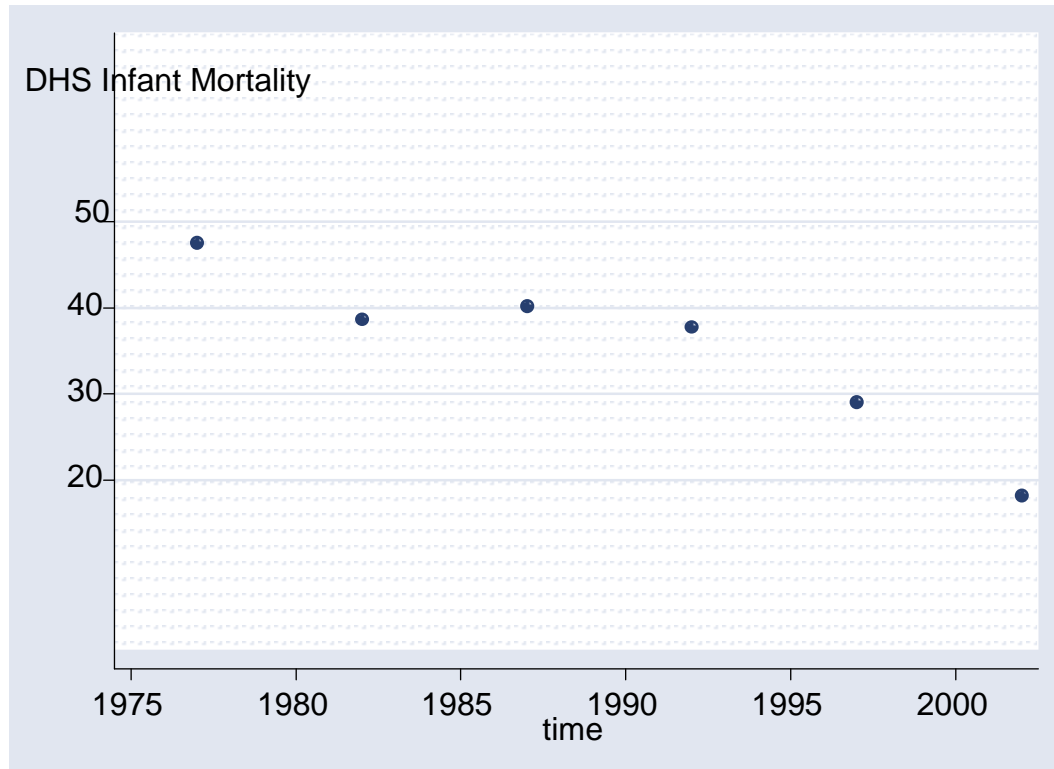
Appendix 11: Durbin's M test for Autocorrelation⁸⁵

	WDI	WDI	WDI	DHS	DHS	DHS	DHS	DHS
	Nutrition	Infant	Under-five	Neonatal	Post Neonatal	Infant	Child	Under-five
	Ui	Ui	Ui	Ui	Ui	Ui	Ui	Ui
Lag Ui	0.966***	1.025***	0.983***	0.796***	1.014***	0.858***	0.920***	0.917***
GDP Per Capita, PPP (current US\$)	-0.0558	-0.0597	-0.0516	0.000660	0.0779	0.0351	0.0431	0.0413
Gini Index	-0.234	-0.139	-0.182	-0.244	-0.350	-0.290	-0.926*	-0.483
Ratio of girls to boys in primary and secondary education	-0.282	-0.323	-0.410	0.177	-1.262**	-0.442	-1.174*	-0.696
1989/1994Time dummy	2.789	2.542*	3.090**	0.135	6.509***	2.869	8.606***	4.722*
2000/2004Time dummy	2.587	2.469*	2.966**	0.146	6.423**	2.840	8.416***	4.650*
Observations	42	74	74	32	33	33	33	33
Adjusted R-Squared	0.867	0.770	0.737	0.354	0.690	0.486	0.673	0.557
Key	** p<0.01	*** p<0.001"						
* p<0.05								
** p<0.01								
*** p<0.001"								

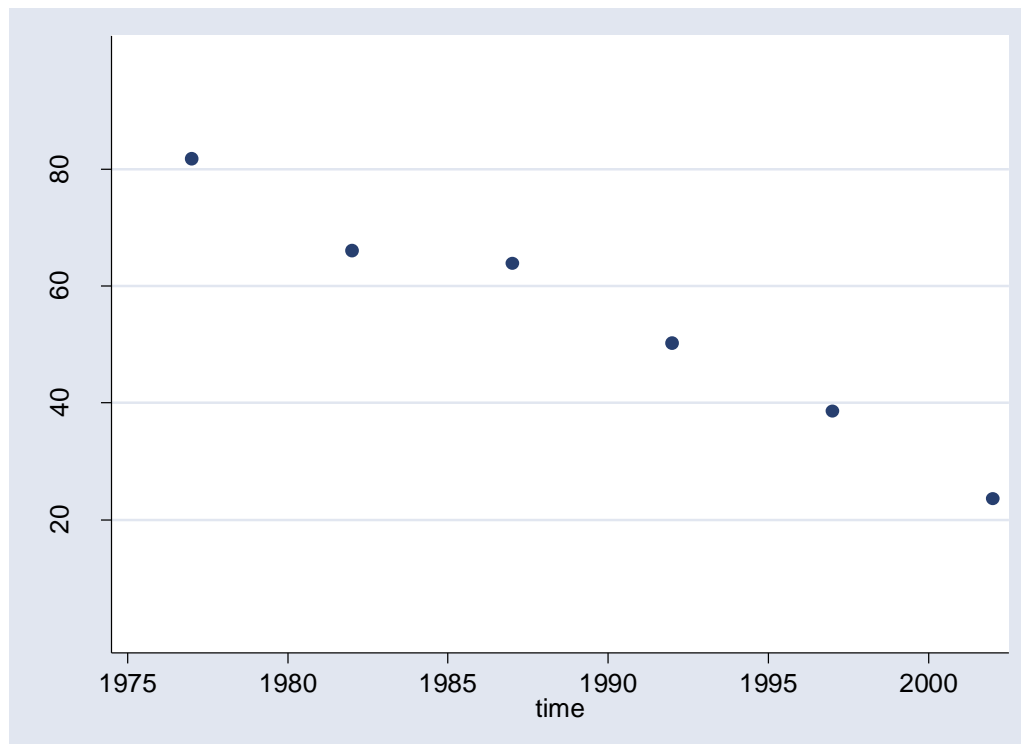
⁸⁵ Autocorrelation was confirmed using the XTSERIAL test.

Appendix 12: Additional Graphs for Part 2

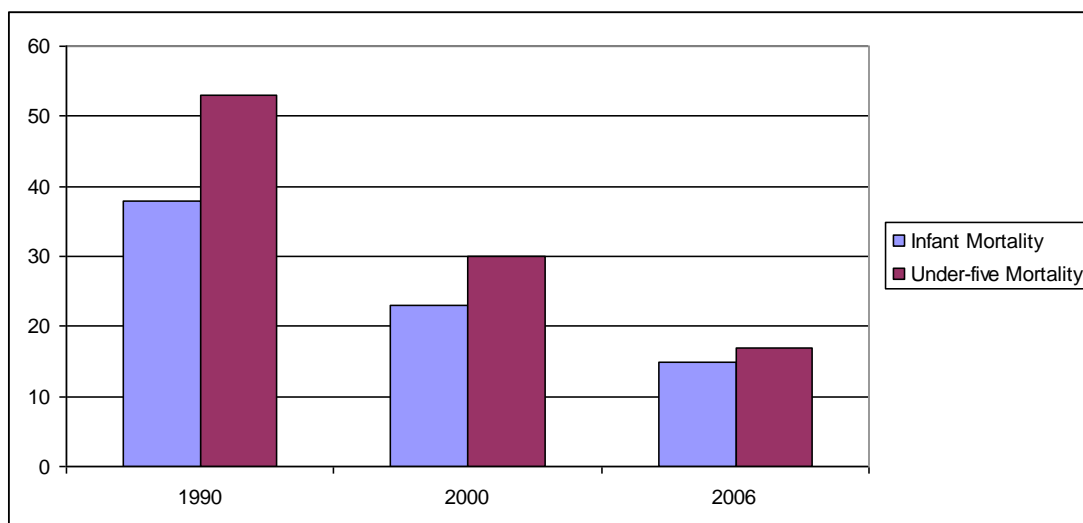
Graph 1: Infant Mortality, Amended DHS Data



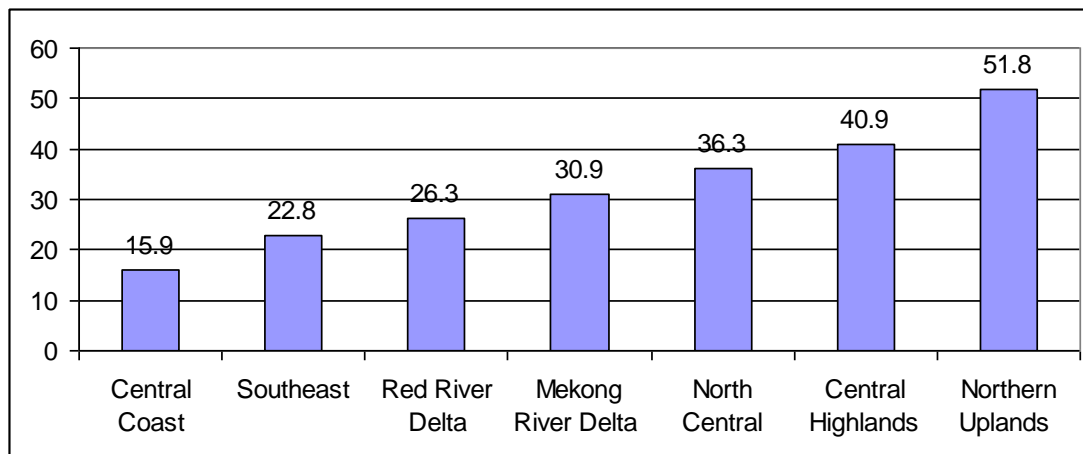
Graph 2: Under-five Mortality, Amended DHS Data



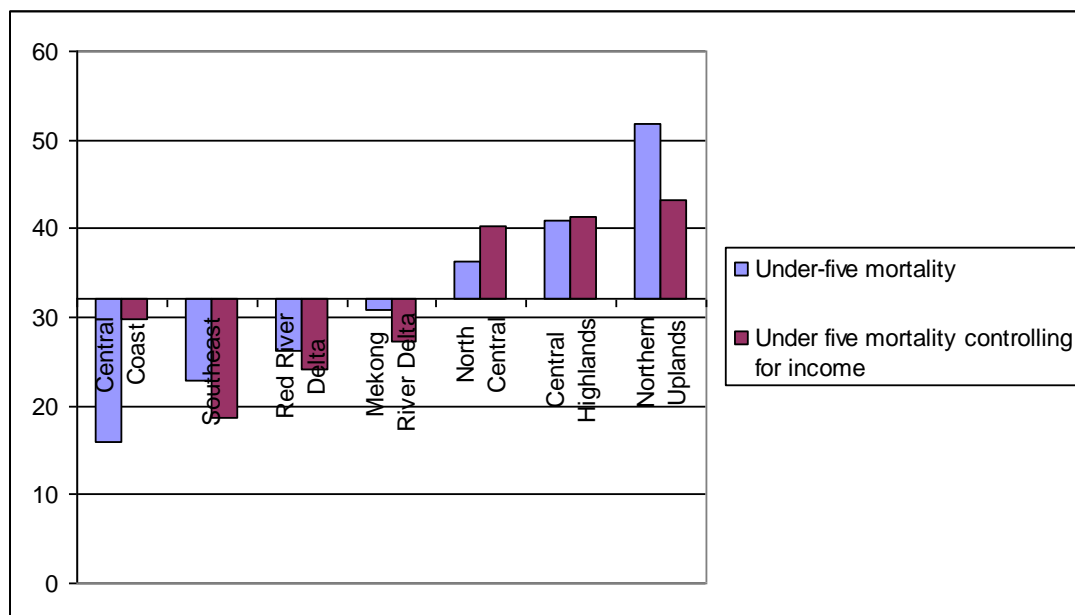
Graph 3: Infant and Under-five Mortality, WHO Data



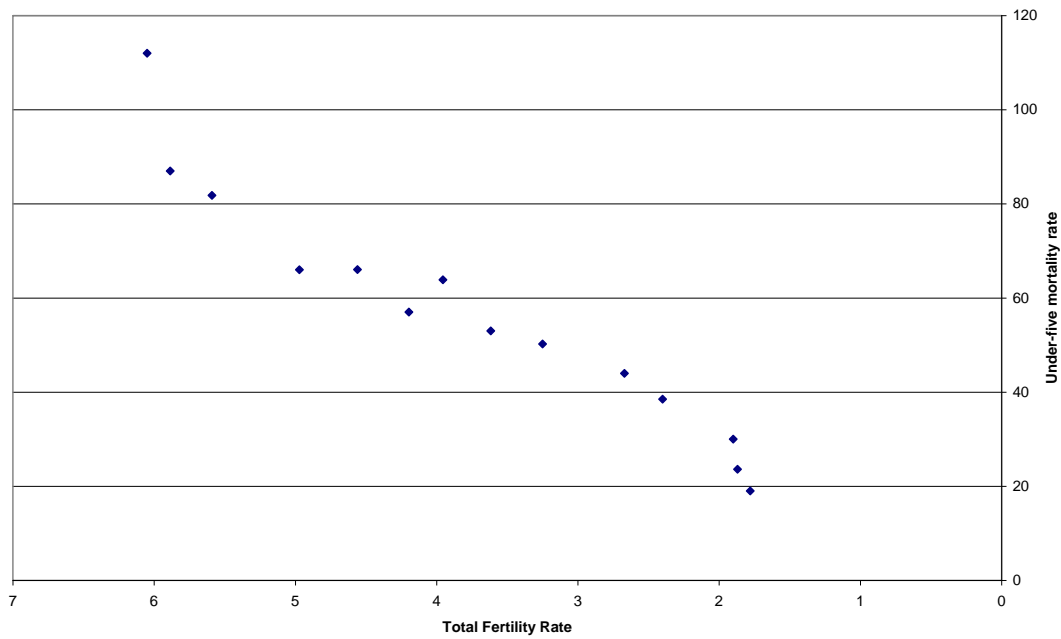
Graph 4: Under-five Mortality in Vietnam by Region



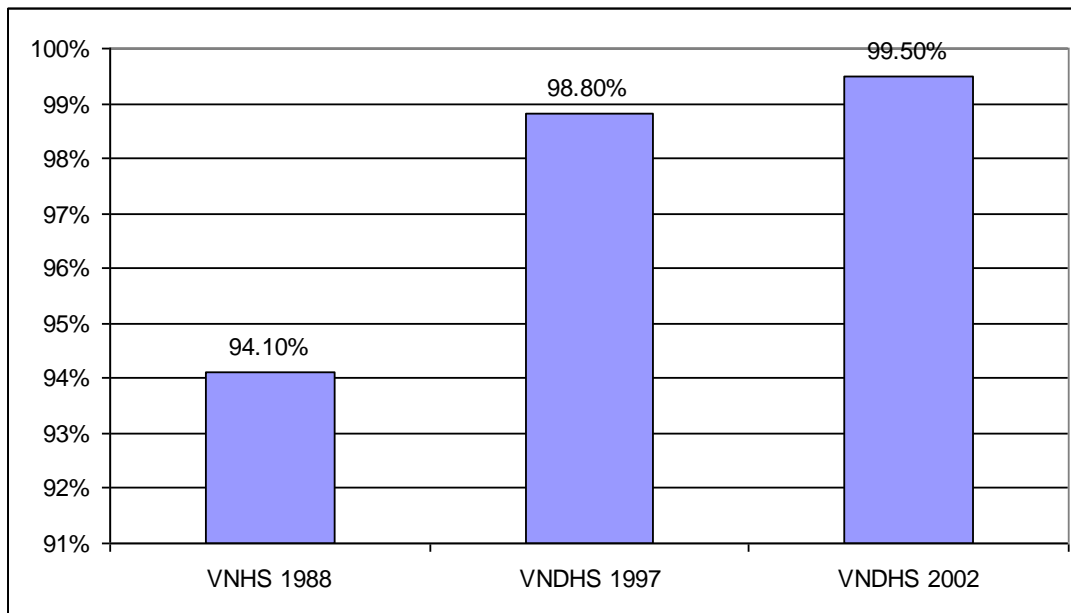
Graph 5: Regional Mortality (X axis is formatted around the average)



Graph 6: Total Fertility and Under-five Mortality

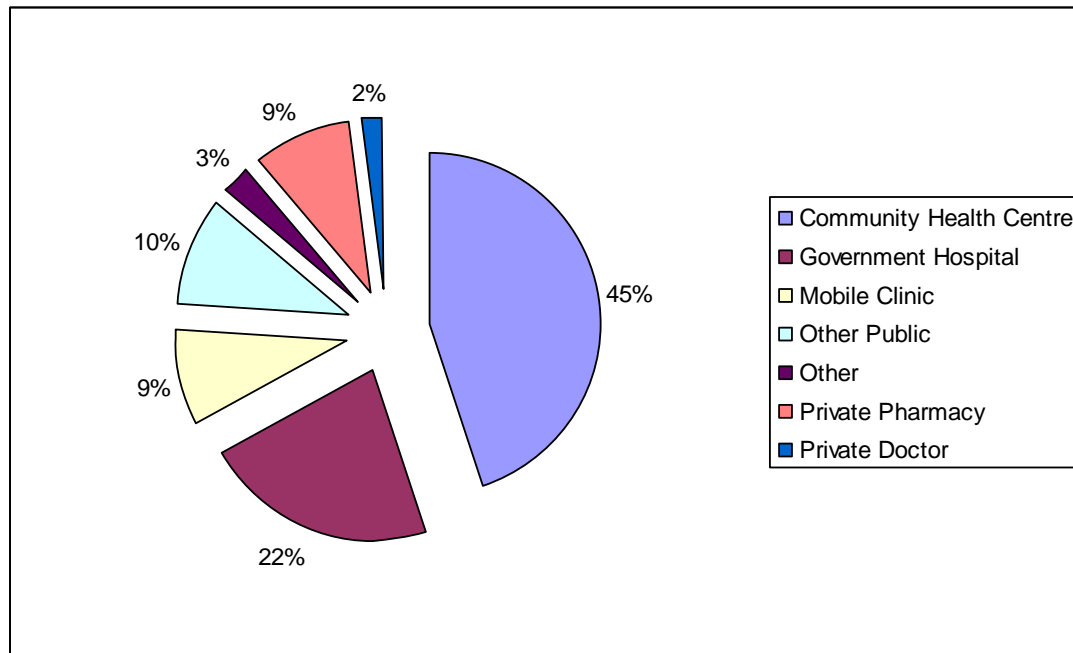


Graph 7: Percentage of Ever-married Vietnamese Women who Know of at Least One Modern Method of Contraceptive⁸⁶

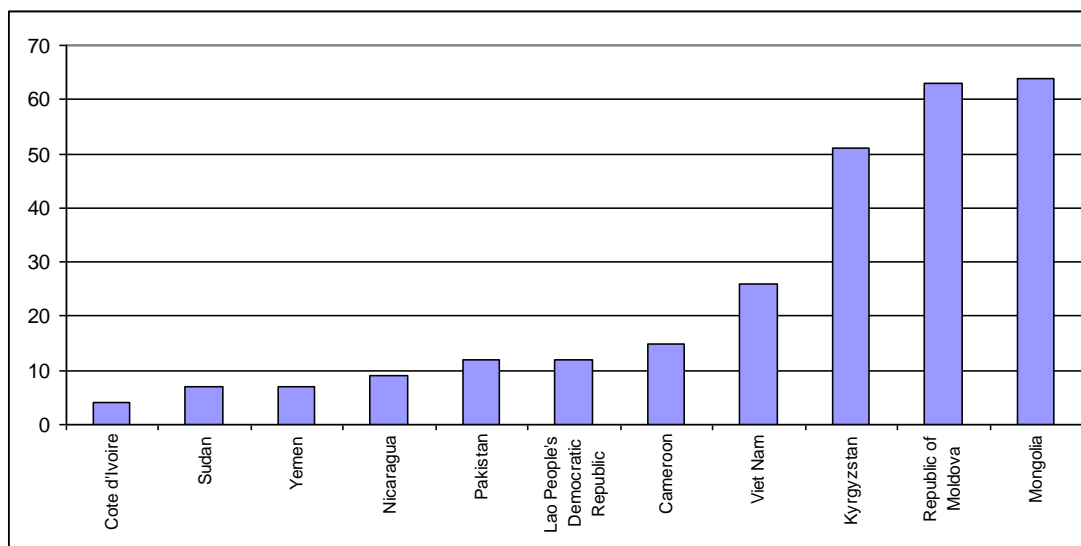


⁸⁶ Adapted from Vietnam's DHS (2002).

Graph 8: Sources of Family Planning Provision among Current Users of Modern Contraceptive Methods⁸⁷



Graph 9: Hospital Beds per 10,000 of the Population in Vietnam and Comparable Countries⁸⁸

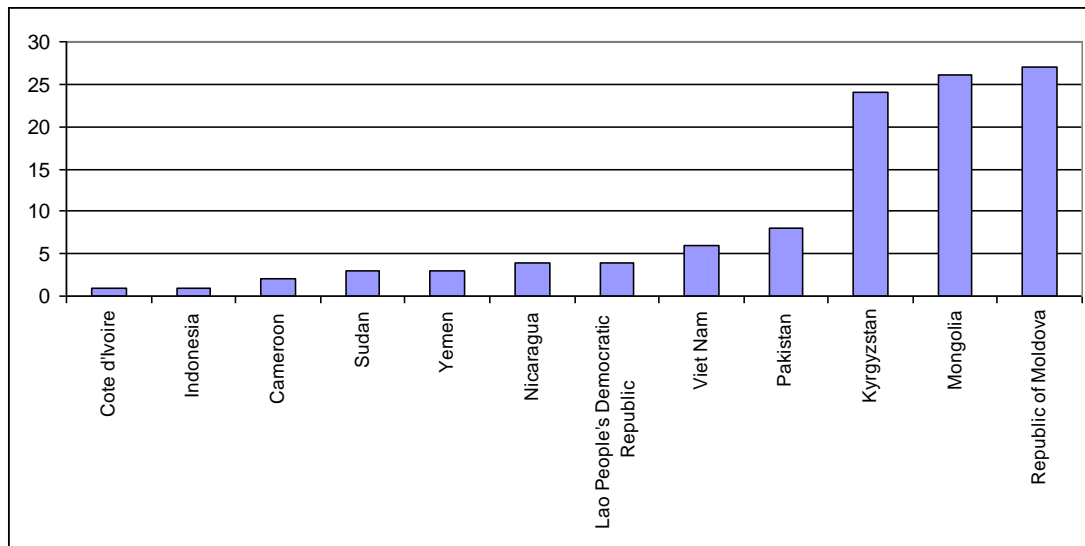


⁸⁷ Adapted from the Vietnam 2002 DHS, page 50.

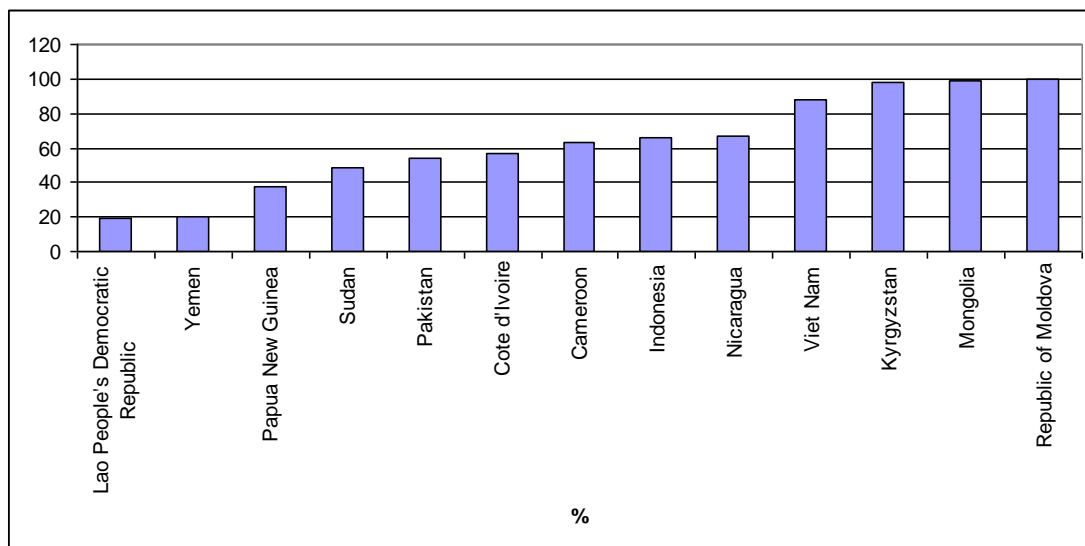
⁸⁸ All countries selected have a per Capita GDP of between 75% and 125% of Vietnam's in 2005. All data are from the most recent year available, which for all countries is between 2000 and 2006. All figures taken from WHO database see:

[http://www.who.int/whosis/data/Search.jsp?indicators=\[Indicator\].\[MBD\].Members](http://www.who.int/whosis/data/Search.jsp?indicators=[Indicator].[MBD].Members)

Graph 10: Physicians per 10,000 of the Population in Vietnam and Comparable Countries⁸⁹



Graph 11: Births Attended by Skilled Health Personnel in Vietnam and Comparable Countries⁹⁰



⁸⁹ See preceding footnote.

⁹⁰ See preceding footnote.

Table 1: Concentration Curves for Child Immunizations

Country	Year	Concentration Curve Value	Standard Error
Yemen	1997	0.36167	0.01811
Cote d'Ivoire	1994	0.21685	0.01623
Pakistan	1990/1991	0.20638	0.02326
Mauritania	2000/2001	0.20121	0.02151
Indonesia	2002/2003	0.12733	0.00942
Vietnam	2002	0.11756	0.01745
Cameroon	2004	0.08658	0.01427
Vietnam	1997	0.06484	0.0229
Nicaragua	2001	0.04391	0.00996
Kyrgyzstan	1997	0.00065	0.01898
Uzbekistan	1996	-0.01238	0.01428

Appendix 13: Are Developing Countries Currently on Track to Achieve MDG4?

Murray et al (2007) have analyzed whether countries are on course to achieve MD4. They predict trends in under-five mortality for nearly all developing countries. Their conclusion is that only 28 middle-income and four low-income countries have a greater than 75% chance of achieving MDG4, and that 37 middle and 44 low-income countries have a less than 20% chance of meeting this goal. You et al (2010) undertake a more recent analysis. They estimate that of the 67 countries with the highest child mortality rates only 10 are on course to have reduced mortality by two thirds between 1990 and 2015.

The above country level studies offer a useful insight into whether countries will achieve MDG4. However, both studies are based on data from international datasets, which may be inaccurate and in some cases is based on interpolations and extrapolations⁹¹. We, therefore, conduct our own empirical analysis of rates of decline in infant and child mortality data using our DHSadj data. In our DHSadj dataset child mortality data refers to whether or not a child died in the first three years of life.

We then calculated, using the OLS regression technique, the trend in infant and child mortality for 61 countries. In this regression our dependent variable was infant or child mortality and the independent variable was the year. Based on this regression we predicted countries' mortality rates for 1990 and 2015. Countries were classified as on track to achieve MDG4 if they were predicted to see a decline in mortality of more than two thirds between 1990 and 2015 and this downward trend was significant at the 10% level. Appendix 3 contains the detailed results for each country level regression. These results show that based on current trends only 22 (36%) and 25 (41%) of the 61 countries analyzed will reduce child and infant mortality respectively by two thirds or more between 1990 and 2015.

This analysis may, however, overestimate the number of countries achieving MDG4 because it used a linear specification. This specification means that mortality must decline or increase at a constant rate. Yet it may be the case that as mortality declines the rate of this decline decreases because it is easier for mortality to decline from 100 to 90 per thousand than from 10 to zero per thousand. Using a linear-log or log-linear functional form allows mortality to decline at a decreasing rate.

These results are displayed in the next table. Using the linear-log functional form, only two and one countries are on course to achieve MDG4 for child and infant mortality respectively. Using the log-linear functional form no countries are currently on course to achieve MDG4.

In summary our results and previous research show that most countries are not on course to achieve MDG4. That is, they will not have achieved a two thirds reduction in under-five mortality between 1990 and 2015.

⁹¹ Murray et al (2007) discuss the problems with the current international databases in some detail.

Table 1: Are Countries on Course to Achieve MDG4?

Number of Countries that will Achieve MDG4 with $P < 0.1$				
		No	Yes	Grand Total
Linear	Child Mortality Rate	39	22	61
	Infant Mortality Rate	36	25	61
Lin-Log	Child Mortality Rate	59	2	61
	Infant Mortality Rate	60	1	61
Log - Lin	Child Mortality Rate	61		61
	Infant Mortality Rate	61		61

Appendix 14: Was Vietnam a Weak or Strong State in the 1980s and Early 1990s?

Fford and Vylder (1996) have argued that Vietnam was a weak state. Their argument is that the ability of families and firms to engage in quasi-legal market activities in the early 1980s, while the government was officially committed to implementing socialism through a planned economic system, demonstrates the weakness of the government. This argument warrants detailed examination because it appears to directly contradict the line taken throughout this thesis that the Government of Vietnam had the power to repress, cooperate with and provide services to its population.

Economic agents engaging in market activities only demonstrates the weakness of the state if the government is committed to repressing such behaviour. The ideological statements of the Government of Vietnam and Communist Party of Vietnam during the 1970s and early 1980s would certainly suggest that the state was committed to restricting the role of markets and implementing socialism. Yet the actions and policies of the government paint a more complex picture. Many of the policies introduced in the late 1970s and early 1980s legalized existing, and encouraged new, market activities, and at times in the 1980s the government recognized the economic rationale and macroeconomic benefits of such policies. The government was then torn between its stated commitment to socialism and the potential benefits of allowing families and firms to engage in more market-oriented activities: it was not straining every sinew in an attempt to control such behaviour. This is most clearly shown by the state's decision not to use military force or its network of informants to stop quasi-legal market activities.

The extent to which the state genuinely wanted to repress these informal fence breaking activities also needs to be evaluated with reference to the threat they posed to the state and those socioeconomic groups upon which the state relied. Informal activities do represent a challenge to the state's ability to control citizens' actions and behaviour; that is to its repressive power. Such informal activities, however, also benefited many of the state's key supporters. There is much evidence for instance that cadres in rural areas economically benefited from such quasi-legal trade activities because they were able to play the role of middleman between city based traders and families in cooperatives who wanted to sell their output. There is also much evidence that the managers and employees of state owned enterprises benefited from the ability of their firms to engage in commercial activities through the market place. There is some evidence that money made through market activities was passed up through the Communist Party enriching civil servants and party members in high level positions. The extent to which the state was committed to stamping out activities which benefits its core supporters can then be questioned.

The Government of Vietnam was of course "weak" in that it lacked the strength to implement a successful planned economy. Yet according to this rationale there has never in the history of the world been a strong state. For history is replete with governments that have attempted to implement a planned economy and have ultimately failed and abandoned such attempts. If a relatively rich super power such as the USSR cannot successfully implement a planned economy in the long term, can we fairly label a poor developing country recovering from a brutal civil war as a weak state because it fails to successfully implement such policies?

Fford and Vylder (1996) also fail to differentiate between types of state power. Even if the Government of Vietnam was too weak to force its citizens not to undertake market-oriented

activities, that is even if its repressive power was low, this does not mean that it was too weak to provide its citizens with services they wanted or to cooperate with its citizens.